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THE PRINCIPLES AND PRACTICE OF BONE CONDUCTION AUDIOMETRY:

A Review of the Present Position.*

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Whereas the technique of air conduction audiometry is well established, the basic principles of bone conduction audiometry are still far from being generally understood. In consequence, the tests are still too often inefficiently performed and hence fail to provide the vital diagnostic information which is their purpose.

As will be shown, this disappointing state of affairs is quite unnecessary, and requires for its correction no more than a rational standardization of our technical equipment and test procedures, whereby proper use is made of the considerable body of acoustic and physiological knowledge now at our disposal.

An essential requirement with all bone conduction tests is the exclusion of the untested ear by means of an efficient masking sound, so that all threshold readings can be validly related to the tested ear. In order to understand how this requirement arises, we need to know first certain facts concerning the transmission of air and bone conducted sounds across the head; second, the physical characteristics of a

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sound which confer upon it maximum efficiency for the masking of pure tones of specified frequency.

The facts relating to sound transmission across the head are conveniently studied in subjects with normal hearing in one ear and complete deafness of the other.

The results obtained are shown in Fig. 1. The calibrated

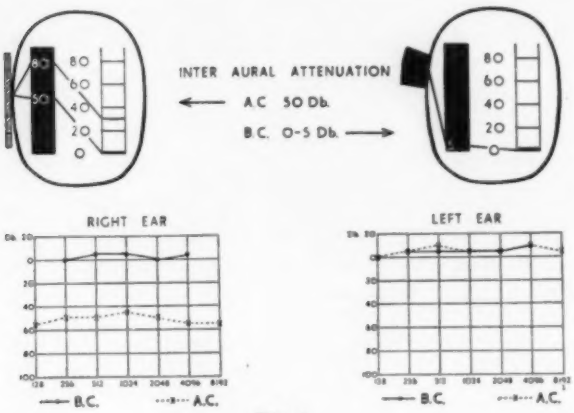

Fig. 1.

columns are taken to represent the available hearing capacity of each ear; the solid column at the left represents total deafness of the right ear. Positive responses to the test tones of different levels above normal threshold are indicated in this and succeeding figures by serrated lines. If an air-conducted sound of 50 db is applied to the right, or deaf ear, it will be heard in the left ear at threshold intensity. Similarly, a sound of 80 db applied to the right ear will be heard in the left ear at a level some 30 db above threshold. The reason for this is that sound from an air conduction receiver is transmitted across the skull with an interaural attentuation of the order of 50 db. By contrast, the interaural attentuation for sound from a bone conduction receiver applied to the right mastoid is negligible, and the sound will be heard at the left ear with a loss of only a few decibels.

In the lower half of Fig. 1 are shown the pure tone audiograms of the right and left ears for air and bone conduction carried out without masking. They indicate normal hearing for the left ear, and a severe conductive deafness for the right ear. In agreement with this, the classical tuning fork tests applied to the right ear lead us into that well-known pitfall of clinical practice, the false negative Rinné; in fact, for the reasons given, the threshold readings, both for air and bone conduction, obtained for the right ear and shown in the audiograms, are false readings derived from the left or untested ear. In the case of air conduction, owing to the comparatively high interaural attentuation of 50 db, such false readings are obtained only when the stimulus intensity exceeds this level. Clearly enough, true readings would result with any degree of deafness of the right ear below this level; hence the rule that when the difference in the air-conduction thresholds of the two ears exceeds 50 db the readings for the worst ear cannot be relied upon, unless the better ear is excluded by masking.

In the case of bone conduction, however, the situation is much more difficult. Here the interaural attenuation is negligible, and hence, if the bone-conduction threshold of the right ear be raised by any amount above 5 db, a receiver on the right mastoid will give false normal readings derived from the opposite untested ear; hence the rule that *all* bone conduction tests require the exclusion by an air-conducted masking sound of the untested ear. A variety of masking noises has been devised to serve this purpose. A few, like those described by Zwislocki (1951), or Denes and Naunton (1952), are efficient, but of the majority it can only be said that they raise the threshold of the masked ear in some vague unspecified manner.

In order to understand what makes a masking sound efficient, it is helpful to consider first the masking of pure tones by white noise, that is to say, noise having a flat frequency spectrum covering the whole audible frequency range with equal sound energy per cycle. In Fig. 2 is shown the elevation of the threshold of a pure tone of 1,000 cycles in

the presence of white noise. The noise intensity is expressed in decibels above normal threshold, and it will be seen that below the 20 db level it produces negligible masking of the pure tone. Above this level there is an incremental equivalence in decibels of masking and of masking noise intensity. At any masking level, therefore, the loudness of the white

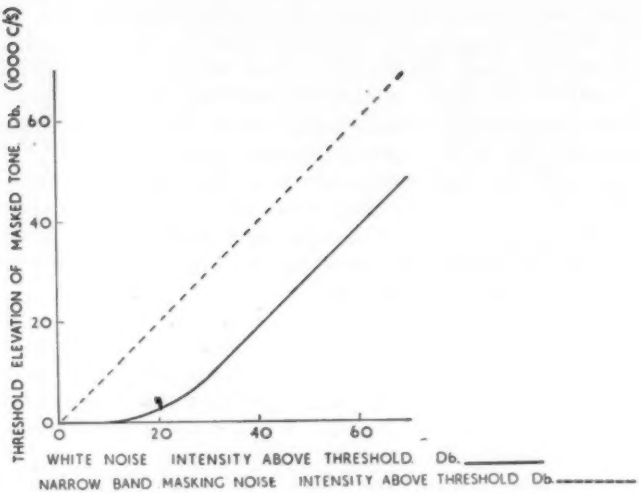

Fig. 2.

noise will be much in excess of the pure tone, since its intensity will at all times be some 20 db higher.

This brings us to the first important requirement of a masking noise, namely, high masking efficiency which Denes and Naunton have defined as "good masking ability, but minimal loudness." Clearly, white noise does not conform to this ideal; nevertheless, it is superior to some of the masking noises incorporated in many commercial audiometers. A typical example is shown in Fig. 3. The masking noise is modified mains hum, its intensity level in decibels above

normal threshold is shown on the horizontal scale, and on the vertical scale the resulting threshold elevation of various pure tones. It will be seen that for frequencies above 1,000 cycles masking is grossly inefficient. At 4,000 cycles no masking at all is produced below an intensity level of some 50 db at which point it is already becoming unpleasantly loud to the subject.

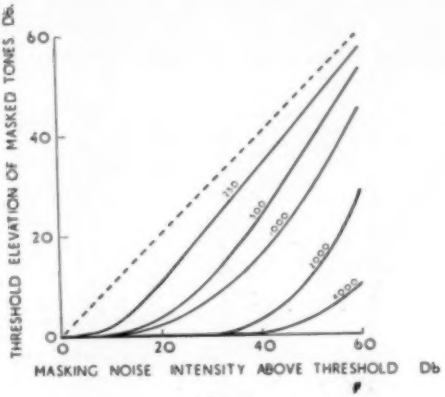

Fig. 3.

Having considered these examples of inefficient masking sounds, we will consider next how we set about our task of designing what we really require, that is to say, sounds which have the maximum possible efficiency for the masking of the limited range of specified pure tones, which are conventionally used for bone-conduction audiometry. Here, as usual, we turn for guidance to some of Fletcher's (1940) early work upon frequency discrimination, loudness and masking, which led him to define the existence of certain so-called critical frequency bands. Their significance lies in the fact, first, that in any masking noise only those frequencies within a certain critical band centered around the pure tone contribute to its masking, and second, when the tone is just audible against the background of noise, the total acoustic power of the components within the band is the same as that

of the pure tone. The principles are illustrated schematically in Fig. 4. This shows noise bands of varying widths centered about a single pure tone which is just audible in their presence.

The acoustic energy, both in the bands and in the pure tones, is represented by the enclosed area of each. Band A is only three cycles wide, and since its total energy is the same as that of the pure tone, the energy per cycle of the noise is only one-third that of the pure tone. Band B is five cycles wide. Once again, its total energy is the same as that of the pure tone, which thus has five times as much energy as each cycle of the band. The width of Band C is that of the critical

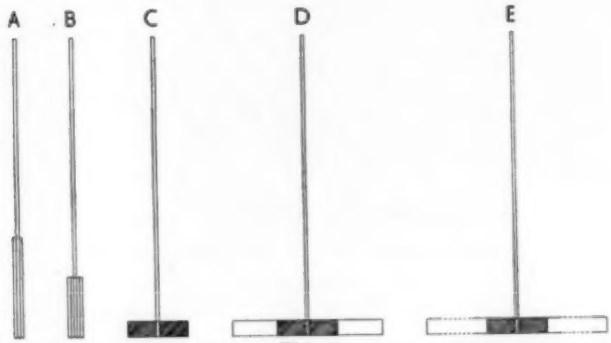

Fig. 4.

band, and yet again the total energy in the band is equal to that of the pure tone.

In bands D and C the noise spectrum extends beyond the critical band, so that the total energy of the bands is much increased. Since, however, it is only the energy in the critical band which contributes to the masking, these noise bands possess no greater masking power, and the energy level of the just masked pure tone remains the same. In other words, those components outside the critical band, while they may have the effect of making the noise very much louder, contribute nothing to its effective masking power. This provides us with the theoretical basis for the concept of high masking

efficiency; clearly our first objective must be to remove as much as possible of these unnecessary components.

Are we likely to gain anything from the use of band widths which are only a fraction of the critical band, as for example in Band B? Apart from the fact that to do so would present very difficult technical problems of sound filtering, we should gain nothing in efficiency and, in addition, would be faced with the added difficulty of the tonal similarity of the pure tone and the masking noise which might result in some con-

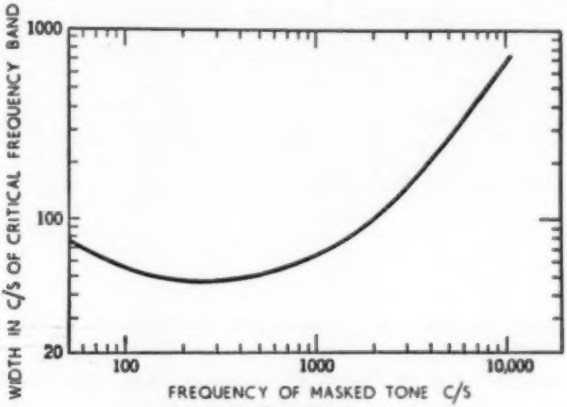

Fig. 5.

fusion to the patient. If, however, the band width of the masking noise is extended to correspond roughly to that of the critical band, as in Band C, then we find that though masking sound and pure tone are identical in pitch, they are still readily distinguishable, and hence are not confused by the subject tested.

The critical band widths for the various frequencies are shown in Fig. 5. As will be seen, they are of the order of only a hundred cycles wide. It would be technically impossible to produce noise bands to these specifications, nor, as will be shown, is this necessary.

In Fig. 6 is shown schematically the equipment we have used for obtaining narrow bands of noise, having the masking efficiency required. A white noise generator is followed in turn by a number of filters of high selectivity centered around each of the pure tones normally used in bone conduction audiometry, followed in turn by an attenuator calibrated in steps of 10 db. Each of the filters can be selected by means of a switch, and passes only a very narrow band of frequencies.

The selectivity curve for the band centered at 1,000 c/s, which is typical of the other curves, is shown in Fig. 7. At

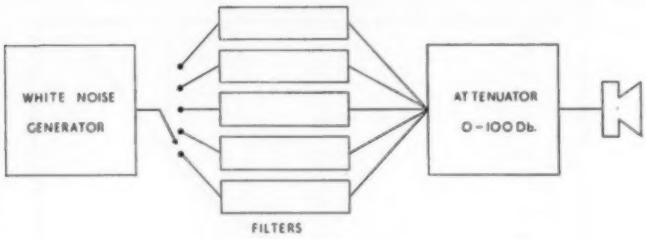

Fig. 6.

the center, the two vertical lines give the measure of the width of the critical band. The curves extend somewhat beyond the critical band at the base; nevertheless, the maximum energy in the band is centered within the two parallel lines.

The dotted line in Fig. 2 illustrates the high masking efficiency obtained with this band.

In contrast to the masking effect of white noise, there is 100 per cent masking efficiency. The curve begins at zero, and there is an exact one-to-one relationship between noise level and masked threshold. Thus, if the noise is raised 20 db, the pure-tone threshold is raised by an identical amount, and so on. All the noise bands are similar in this respect, and we regard it as one of their most important features. Thus, if it is desired to raise the threshold of a pure tone by, say 40 db, it becomes a simple matter of raising the intensity level of the noise band by exactly the same amount. The

practical importance of this is best illustrated by considering the simple application of these noise bands to the technique of bone conduction tests.

Fig. 8 is a schematic illustration of the rationale of the procedure employed at the Otological Research Unit, and referred to for convenience as the "shadowing" technique. Let there be a pure nerve deafness of 40 db of the subject's right

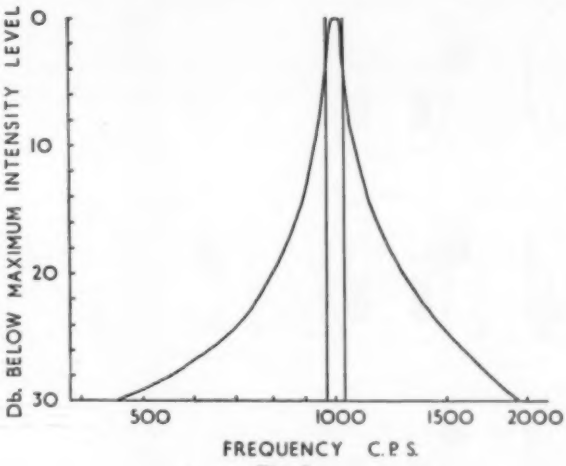

Fig. 7.

ear to be tested, and no loss of hearing of the left ear; as before, the columns of each side are taken to represent the available hearing, and are calibrated in decibels. The solid portion of the left column represents the nerve deafness of the right ear. A bone conducted sound at normal threshold intensity applied to the right mastoid is heard in the subject's left ear. This provides us with the first point on the graph shown below, in which the masking intensity at the left ear is plotted on the horizontal scale and the bone-conduction threshold responses on the vertical scale.

Now, as shown in Fig. 8, the masking noise at a level of 10 db applied to the left ear raises its threshold for the bone

conducted test tone by 10 db. In the same way, increases in masking noise intensity of 20 or 30 db will bring about increases of identical magnitude in the threshold values for the test tone. Reference to the graph shows, up to this point, this step by step correlation, the "shadowing" effect between the intensities of masking noise and test tone thresholds. When, however, the masking noise reaches the 40 db level it

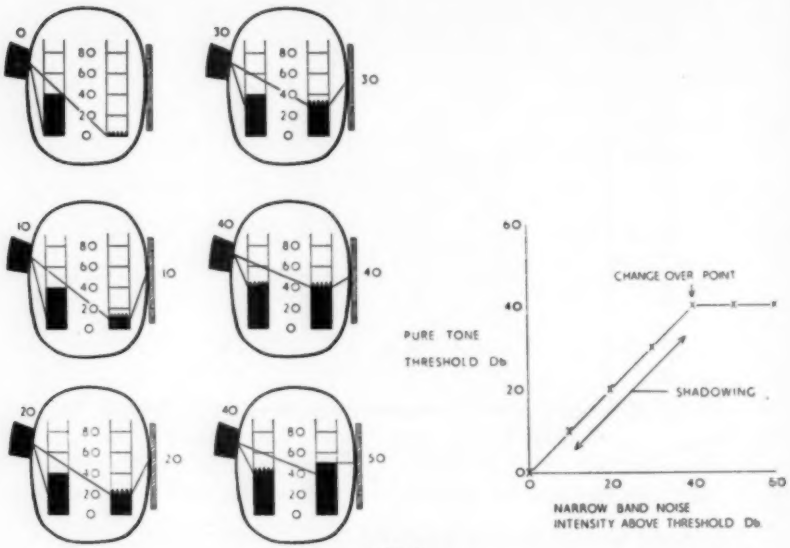

Fig. 8.

will be seen that the bone conducted test tone will be heard at threshold by the right as well as the left ear. A further intensity increase of the masking noise to 50 or 60 db will further raise the threshold of the left ear alone, leaving the test tone to be heard by the right ear alone without further elevation above its true threshold value, 40 db. The true threshold for the right ear will thus be shown at a point on the graph, referred to as the "change-over" point, at which the slope of the curve changes from 45 degrees and becomes horizontal. In the case being considered, the change-over

point occurs at 40 db. If, extending the argument, the deafness of the tested ear had been 50, 60 or 80 db identical values of masking noise intensity would have been required to reach the "change-over" points. In all cases, true threshold readings would be obtained.

The essential steps of the test procedure are thus:

1. The demonstration of the "shadowing" effect. This establishes that both masking noise and test tone are confined to the untested ear.

2. The identification of the "change-over" point. This establishes the true threshold of the tested ear.

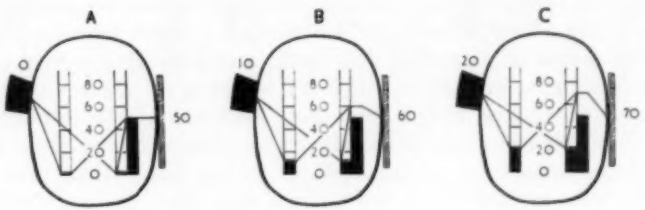

Fig. 9.

The case analyzed in Fig. 8 is one of comparative simplicity in that the unilateral deafness was of the "nerve" type. In unilateral deafness of the conductive type, however, we are confronted with a situation of considerably greater difficulty. This arises, as will be shown, from the fact that the interaural attentuation of an air-conducted masking sound does not exceed some 50 db.

The situation is illustrated in Fig. 9.

Here, the subject has normal hearing of the right, or tested ear, and a pure conductive deafness of 50 db of the left ear. This is indicated by the solid area adjoining the calibrated column which represents the inner ear hearing capacity of the left ear, reduced in B and C by masking.

In ascertaining the bone-conduction threshold of the right ear we must, as usual, apply an air-conducted masking noise to the left ear. Here a serious difficulty arises, since before this noise can become effective its intensity must reach 50 db, the amount of the conductive deafness. At this intensity it will just overcome the interaural attenuation for air-conducted sound, and begin to exert a masking effect upon the right ear as well as the left. If the intensity of the masking noise be further increased in steps of 10 db, a "shadowing" effect will occur, and the bone-conduction thresholds, derived equally from the two ears, will rise *pari passu*. In this instance, however, the "shadowing" effect will continue indefinitely, and no "change-over" point will be reached.

The underlying difficulty is clearly to be found in the fact that the interaural attenuation of the air-conducted masking noise has a limit of some 50 db. What is needed, therefore, is some means of extending this limit, and providing a zone of masking noise intensities which will be above the air-conducted threshold of, and at the same time confined to, the left ear. Within this zone, the "shadowing" procedure could be applied, and the absence of a "shadowing" effect would then prove that bone-conduction threshold readings were derived from the right ear, and could be accepted as valid.

The manner in which such an increase of the interaural attenuation can be accomplished stems from a brief report on the use of insert receivers which appeared in the Report of the Electro-Acoustics Committee "Hearing Aids and Audiometer," 1947. In this it was suggested that the acoustic linkage between the two ears which occurs when any air conduction receiver is used arises from the vibrations imparted to the skull at the air-bone interface bounding the receiver. Both Littler, et al. (1952) and Zwislocki (1953) have carried out further studies, and it has been shown that the vital factor affecting the attenuation is the area of the head exposed to the air conducted sound waves. Thus, the smaller the area the greater the attenuation, and in the limiting case when an insert receiver is used, thus presenting the smallest possible surface to the skull, the interaural attenuation increases to a figure as high as 90 db.

As Littler, et al. (1952) so ably pointed out, the practical importance of this finding is considerable, making possible as it does the accurate determination of bone-conduction thresholds in the presence of conductive deafness well beyond the limit of 50 db. This is illustrated in Fig. 10. Masking noises of intensities up to 90 db are applied to the left ear by means of a rubber-tipped-insert receiver without fear of cross conduction to the right ear.

Thus, at any intensity between 50 db, the air-conduction threshold of the left ear, and 90 db, the masking noise will be effective in the left ear alone. If we now apply the "shadow-

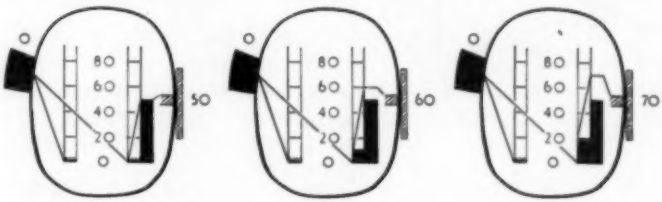

Fig. 10.

ing" procedure, and make step by step changes of masking intensity within this range, they will be found to leave unaltered the bone-conduction threshold readings. In other words, the "shadowing" effect is absent, and the threshold reading, 0 db, can be accepted as valid for the right ear.

When the deafness is bilateral, and of the mixed conductive and nerve type, bone-conduction tests may be greatly complicated, and indeed it is only within certain limits that accurate results are then obtainable.

An audiometrician needs to know what these limits are, and it is fortunately possible to define them with reasonable accuracy. The situation is analyzed in the cases shown in Fig. 11 (A, B and C). In 11-A the subject has a nerve deafness of 60 db of the right ear and conductive deafness of 30 db of the left. In order to establish the bone-conduction threshold of the right ear, the usual masking noise must be

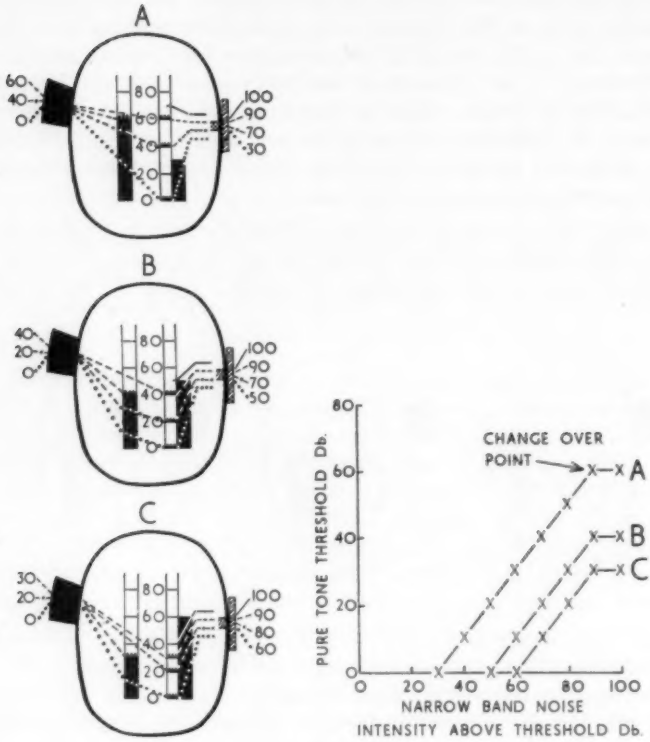

Fig. 11.

applied to the left, and owing to the conductive deafness its minimum effective intensity will be 30 db. Above this point shadowing will occur, as shown in the graph (A); that is to say, both masking noise and test tone will be confined to the left or untested ear. When the masking intensity reaches the 90 db level, its true masking effect upon the left ear will be restricted to 60 db by the conductive deafness and the test tone will now be heard at threshold in the right ear as well as in the left. This is the critical change-over point, and beyond it a further increase of masking intensity to 100 db,

the maximum practicable level, will raise the threshold of the left ear by a further 10 db while leaving unaffected at 60 db that of the right ear. This, therefore, will be its true threshold.

Two factors determine the results thus obtained. First, 100 db is accepted as the maximum intensity of masking noise which it is practicable to use for clinical tests. With this the critical change-over point occurs at a level 10 db lower, 90 db, and to this it is convenient to refer as the maximum critical masking intensity. Second, the threshold of the tested ear can be determined only if it lies at or below the level of masking induced by the maximum critical masking intensity in the untested ear.

Now, in Case 11-A this level is reduced by 30 db, the amount of the conduction deafness and hence, 60 db is the maximum measurable elevation of the bone-conduction threshold of the right ear. From this we may derive the limiting formula:

Maximum measurable bone-conduction threshold=90 – conductive deafness (db) of opposite ear.

The application of the test procedure to the three cases A, B and C, represented in Fig. 11, is shown in the graph.

In all three the amount of the nerve deafness of the tested ear is the maximum measurable according to the limiting formula. Thus:

Case A 60 db (90-30).
Case B 40 db (90-50).
Case C 30 db (90-60).

In each case the true threshold is given by the change-over point which occurs at the maximum critical intensity, 90 db of masking noise.

Although for simplicity of explanation the cases shown in Fig. 11 have been selected to exhibit conductive deafness alone of the untested ear and nerve deafness alone of the tested ear, accurate bone-conduction tests are still possible within the limits of the formula when the deafness, of either

one or both ears, is of the mixed type. This is illustrated in Fig. 12. In the right ear there is a conductive deafness of 40 db with a nerve deafness of 60 db. The air-conduction audiogram thus shows a loss of 100 db. In the left ear there is a conductive deafness of 30 db with a nerve deafness of 50 db. The air-conduction audiogram thus shows a loss of 80 db. With no masking noise applied to the left ear the bone-conduction threshold reading is 50 db, derived, in fact,

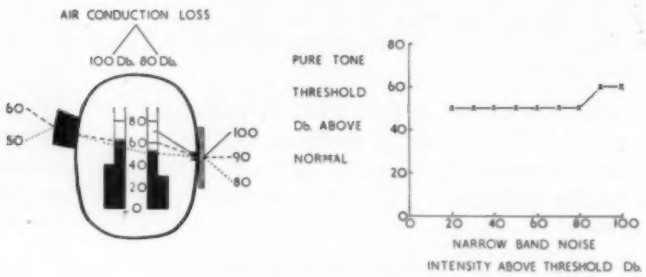

Fig. 12.

from the left ear, though this cannot be specified at this stage of the test.

This reading remains constant with increase of masking noise intensity up to 80 db. With the masking intensity increased to 90 db, however, and still confined at this level to the left ear, the threshold reading is increased to 60 db and remains unchanged when the masking intensity is further increased to 100db. As shown in the graph, "shadowing" occurs between the 80 and 90 db levels of masking noise intensity with the "change-over" point at 90 db. It follows that up to the 80 db level of masking noise intensity, the threshold reading, 50 db, must be derived from the left ear; further, that at and above the 90 db level, the threshold reading, 60 db, is derived from the right ear. Subtraction of these bone-conduction losses from the air-conduction losses gives the amounts of the conductive deafness, thus:

Right ear 100 – 60—40 db.
Left ear 80 – 50—30 db.

In each case, the amount of the nerve deafness is the maximum which can be measured according to this formula. Thus for the right ear:

60 db (amount of nerve deafness) —90 – 30 (amount of conductive deafness of left ear).

For the left ear:

50 db (amount of nerve deafness) —90 – 40 (amount of conductive deafness of right ear). As will be seen from the foregoing analyses, the use of the maximum efficiency masking technique described in this communication brings within the resolving power of air and bone-conduction audiometry all but the few exceptional cases which lie beyond the bounds of the limiting formula.

In all cases, however, careful attention should be given to the fundamental features of the test procedure which is outlined as follows:

1. Establish the air-conduction audiogram of both ears in the normal way with masking, if necessary, of the untested ear, *i.e.*, when the difference in the hearing loss between the two ears exceeds 50 db.

2. Find the bone-conduction threshold with the bone-conductor applied to the mastoid of the tested ear without masking of the untested ear.

3. Apply the masking noise of the appropriate band to the untested ear by means of an insert receiver and find a bone-conduction threshold reading.

4. Apply the "shadowing" procedure thus: Increase the level of the masking noise by 10 db above threshold and retest the bone-conduction threshold. If the bone-conduction threshold is raised by 10 db increase the masking intensity by another 10 db and repeat. Continue this procedure until the point is reached at which the bone conduction remains constant with further additional incremental steps of 10 db

of the masking noise. This is the "change-over" point, and gives the true bone-conduction threshold of the tested ear.

Suggestions for the improvement of standard equipment tend to encounter two kinds of objections: first, that they involve excessive technical difficulties; second, that the solution they offer is far from complete, and hence further development should be awaited.

Neither of these objections appears to be applicable in the present situation. No serious difficulties attend the generation or application of the masking sounds described; and as for further developments, these cannot be expected since in many respects the efficiency obtained is 100 per cent, at which level an ample reserve is provided for the attainment of practically all the clinical results which are required. It is, therefore, to be hoped that the methods described will find useful application in improving the design of standard audiometric equipment.

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THE JAMES E. NEWCOMB AWARD.

Dr. Joel J. Pressman, of Los Angeles, was chosen by the members of the American Laryngological Association at its annual meeting recently, to be the recipient of the James E. Newcomb Award for his outstanding contribution to research in otolaryngology.

FRONTAL SINUS SURGERY.

The Use of Mucoperiosteal Flap for Reconstruction of a Nasofrontal Duct.*

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INTRODUCTION.

Surgery of the frontal sinus has been fraught with failure for one main reason: closure of the nasofrontal duct.[8,12] This paper presents a method of maintaining patency of the nasofrontal duct. The original description was published in the South American literature by Tato[9] in 1948. The method consists of rotation of mucoperiosteal mucous membrane flaps from the nose upward into the frontal sinus to create a new nasofrontal duct common to both sinuses. We present a small series with an advanced form of frontal sinus disease, *i.e.*, unilateral orbital complications, fistulae, mucoceles, and/or bony wall destruction, where the problem of the nasofrontal duct is obvious.

REVIEW OF LITERATURE.

In reviewing the literature on surgery of the frontal sinus it is apparent that closure of the nasofrontal duct is stated to be the greatest cause of recurrence.[12] In 1942 Goodale[5] reviewed 182 cases of frontal sinus disease at Massachusetts Eye and Ear Infirmary and found a recurrence rate of 30.8

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per cent for infection and 22.2 per cent for mucoceles. Prior to his paper and subsequent to this, various methods have been suggested to maintain patency of the duct. These include the use of reamers, dilators, skin grafts, mucosal grafts and the permanent placement of plastic, gold and tantalum tubes. Walsh's[11] suggestion that the nasofrontal duct be left alone, if not damaged by disease, is obviously the best treatment; however, there are many instances where the duct is obliterated or destroyed by disease, but where there is no osteomyelitis. It is for these cases that the mucosal flap method is advocated to reestablish adequate drainage.

At the present time several surgical procedures are available for treating disease of the frontal sinus and the principle underlying each is different. These are:

1. The classic obliterative procedure (Riedel and Lyman).[7]

2a. The osteoplastic flap approach for exposure (Gibson and Walker).[4,6]

2b. The osteoplastic flap procedure with obliteration (Bergara).[1,10]

3. The Lynch type external frontal and its variations.

4. The Ermiro de Lima frontotomy through a trans-antral ethmoidectomy.

5. Simple trephining of the frontal floor.

6. Mucosal flaps turned up from the nose either unilateral (Sewell and Boyden[2,3]) or bilateral (Tato[9]).

Each method has its advantages and disadvantages. The classic obliterative procedure is advocated only for cases of osteomyelitis of the frontal bone; it is usually deforming and is not necessary if alternative methods are available.

The osteoplastic flap gives very wide exposure in dealing with tumors or osteomas of the frontal sinus, but this method does not in itself provide drainage and presumes a functioning nasofrontal duct.

The success of the osteoplastic approach with obliteration

of the frontal sinus cavity with scar tissue depends upon the total obliteration of the sinus and of the nasofrontal duct with removal of all mucous membranes.

The Lynch type external frontal is satisfactory if the region of the nasofrontal duct is not involved by disease. When it is, a new nasofrontal duct is required. This is maintained by the use of artificial tubes for a variable period of time and is not ideal because of the lack of cilia and normal glands; a mucosal-lined duct is preferred. If a large opening is made into the ethmoids this will frequently close in time regardless of initial size.

The De Lima approach to the frontal via the antrum and ethmoids is the procedure of choice for primary disease in antrum and ethmoids with secondary frontal involvement, but this method should not be used to provide permanent drainage when the nasofrontal duct is irreversibly obliterated by the disease. A combination of the Bergara osteoplastic or Lynch type methods with the De Lima approach still does not always assure a permanent drainage from the frontal sinus, in view of the marked tendency to scar formation in the region of the frontal floor which may obliterate the surgically created opening into the ethmoids.

The trephine method is an emergency procedure and is done to relieve the acute purulent infection and provide temporary drainage.

The use of mucosal flaps from the nose is not new. They may be unilateral or bilateral. Actually this method is a means for reconstruction of the new nasofrontal duct combined with an approach to the frontal sinus. The bilateral flap technique is preferred because the new opening is lined on two sides with an absence of a midline partition and is more likely to stay open than one lined only on one side. By turning both flaps the new duct is made wider, and tendency to stenosis is reduced by loss of the midline wall, which in essence is the inferior extension of the intersinus septum.

INDICATION FOR FRONTAL SINUS SURGERY.

A generation ago a great deal of radical sinus surgery was

performed, and irreversible harm was done. Exenteration of the interior of the nose markedly alters the normal physiology and left many people "nasal cripples." Since then there has come a better understanding of the normal function of the nose and a more conservative approach to sinus problems. Whenever possible normal structures are left undisturbed and mucosa which has not suffered irreversible changes due to infection is left intact. The main objective has been to achieve good drainage. When this is established much of the pathology will clear up without radical surgery. Hence surgical attack on the frontal sinus is indicated primarily for: *1.* orbital complications such as cellulitis, abscess or displacement; *2.* mucocele; *3.* fistula; *4.* chronic severe recurrent frontal sinusitis with severe local signs and symptoms due to infection or associated with osteoma. We assume that the nasofrontal duct must be closed to produce this localized pathology. Some cases require surgery to the antra and ethmoids as well as to the frontals. This is usually done as a separate procedure either before or after the frontal surgery, but it may be combined.

SURGICAL TECHNIQUE.

A curved incision as described by Lynch is made down to and through the periosteum. It begins in the eyebrow and extends medially to a point midway between the bridge of the nose and the inner canthus of the eye. The incision is continued inferiorly along the lateral edge of the nasal bone to its distal edge and then curves laterally two or three millimeters. The periosteum is elevated from the supra-orbital area, the medial wall of the orbit, the nasal bone and the ascending process of the maxilla. The anterior and posterior ethmoidal arteries are divided and tied or cauterized. The supra-orbital nerve and vessels are sacrificed. The trochlea and the lacrimal sac are detached and displaced laterally with the globe. The frontal sinus can then be entered either through the floor or via the anterior ethmoid cells.

Frequently the disease process has ruptured through the frontal floor and the sinus is entered while making the in-

cision. In many cases the entire roof and medial wall of the orbit have been eroded, and the frontal and ethmoid sinuses are one large cavity full of fibrous and granulation tissue. The frontal sinus is entered and the opening enlarged by removing the floor and overhanging supra-orbital bone with rongeurs.

If the ethmoids are not involved, they are not disturbed any more than is necessary for the formation of mucoperiosteal flaps. A complete ethmoidectomy is done at this stage if indicated. When this exenteration of frontal and ethmoid regions is complete the formation of the mucoperiosteal flap is begun.

Using a cutting burr as used in mastoid surgery and curettes, the nasal bone, lacrimal bone and a portion of the ascending process of the maxilla on the involved side are removed (see Fig. 1). The intersinus septum is taken down, and the nasal spine of the frontal bone is thinned. There is a triangular wedge of bone lying between the medial portion of the two frontal sinus cavities and the bridge of the nose. This must be removed and made smooth.

There should now still remain a mass of bone lying anterior to the cribriform plate and just superior to the vault of the nose. This is the root of the perpendicular plate of the ethmoid and the nasal septum (see Fig. 1). This bone is carefully removed until the mucous membrane of the nasal vault is exposed. Bone is removed anteriorly as far as possible without lowering the projection of the dorsum in order to give as much antero-posterior dimension as possible to the mucosal flap. When one side has been exposed further bone is removed to expose the vault on the opposite side. The width of the flap is limited anteriorly by the midline projection of the nose and posteriorly by the anterior end of the cribriform plate (see Fig. 1). The flaps are then pushed down into the nose, and the bony septum is removed with burr and rongeurs. Great care must be taken not to tear the flaps. When the mucosal domes have been well exposed and their future bed fashioned the nearest side is cut first, the two layers are cut with curved scissors as far anteriorly as pos-

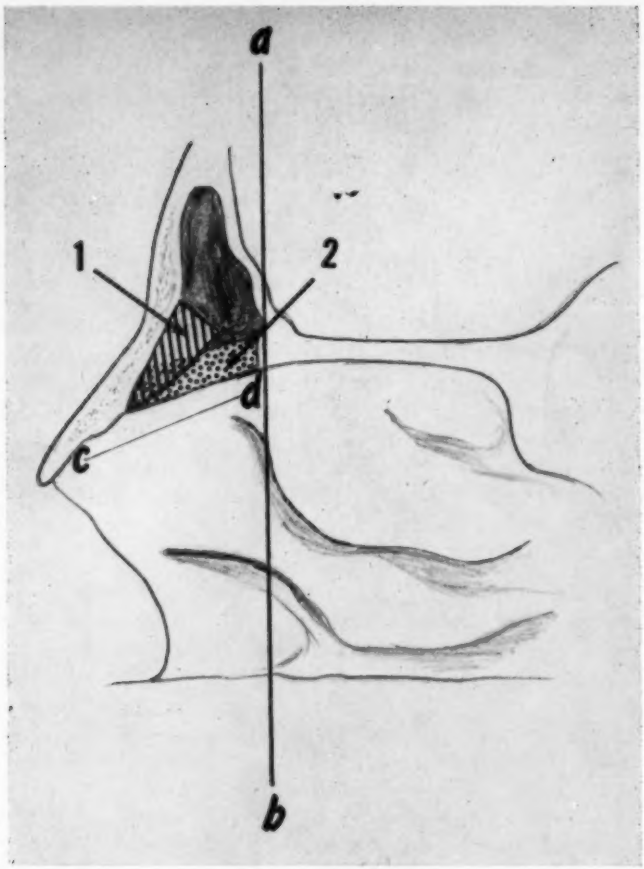

Fig. 1. Sagittal section of frontal sinus. View of right medial wall of frontal sinus. ab—An arbitrary line marked posteriorly by the posterior wall of the frontal sinus. When projected inferiorly this represents the anterior attachment of the middle turbinate. The cribiform plate is posterior to this line. cd—antero-posterior dimension of dome of mucoperiostial flap. 1—heavy wedge of bone deep to glabella. 2—root of perpendicular plate of ethmoid.

sible. The medial layer is cut as far inferiorly as can be reached. A similar incision is made posteriorly, usually sloping downward and forward (see Figs. 1 and 2).

Pressing the double layered flap laterally the mucosal attachment to the septum is cut and the flap turned upward and laterally. Usually a further small portion of the ascending

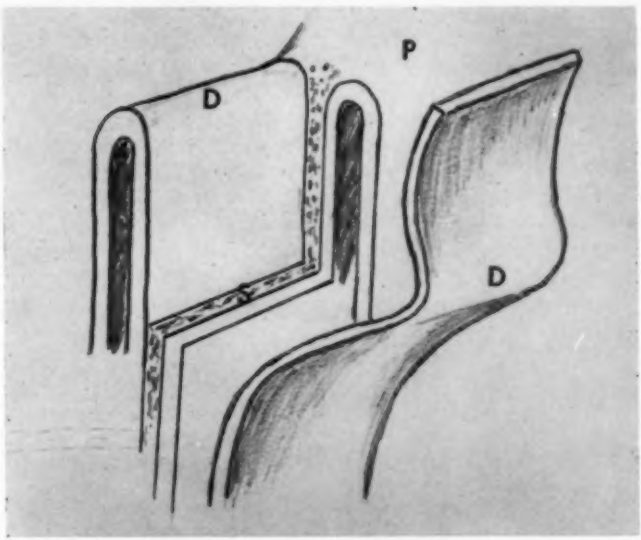

Fig. 2. Preparation of septo-mucoperiostial flaps (modified after Tato). D—dome. S—perpendicular plate of ethmoid and septum partially removed. P—posterior wall of frontal sinus.

process of the maxilla must be removed so that the flap will lie flat on the peri-orbita (see Fig. 3). This flap is carefully tucked out of the way and the opposite flap cut in a similar manner. The area of bone lying between the open field and the opposite nasal bone, trochlea and lacrimal sac, must be smooth and flat so that the mucosal flap can be turned upward and laterally into the opposite frontal sinus (see Fig. 3). We must try to achieve a symmetrically funnel-shaped opening into both frontals draining into the anterior nasal

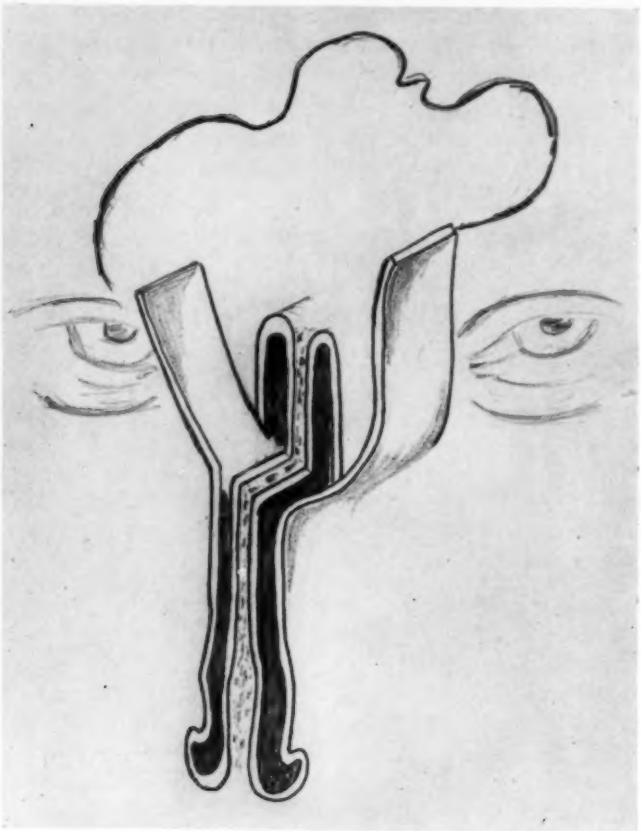

Fig. 3. Final position of bilateral septo-mucoperiostial flaps (modified after Tato).

vault with intact mucous membrane flaps lining both the lateral walls of the funnel. On the involved side the flap is sutured once or twice to the peri-orbita. On the opposite side the flap is just laid into position.

Two large rubber catheters are inserted through the nose, up through the new funnel opening into both frontal sinuses. These help hold the flaps in position. They are also held by

nasal packing and by a heavy silk suture passing through the catheters and over a button on the forehead. This is left in for at least three weeks. The nasal packing can be removed in two to four days.

VARIATION OF TECHNIQUE.

Once the frontal floor has been opened the sinus is examined and the area of the nasofrontal duct inspected. This area is usually markedly involved with granulation tissue and scarring, thus requiring the creation of a new duct. Such cases are frequently bilateral. It is best to remove all the frontal floor to prevent pocket formation laterally.

Van Alyea and others have advised leaving mucous membrane in the sinuses when irreversible changes have not occurred, providing adequate drainage is possible. The mucosa is not completely removed routinely, only that portion showing what is considered irreversible changes, such as intra-mucosal abscesses and large amount of granulation tissue, etc.

We have left inactive diseased mucous membrane, such as mucocele lining over posterior wall erosion, on the assumption that, with a new functioning nasofrontal duct which provides permanent drainage, the mucosa would return to normal, and the follow-up has borne out this assumption.

If the supra-orbital margin has been eroded anteriorly the edges are smoothed with rongeurs. When the posterior wall has been destroyed and the dura is exposed this area is examined for extra-dural abscesses.

The anterior end of the middle turbinate usually marks the posterior extent of the frontal floor in the midline. Hence in preparation of the mucosal flaps dissection anterior to this point clears the cribriform plate (see Fig. 1). The anterior ethmoids only are entered in fashioning the bilateral flaps. A complete ethmoidectomy is not routinely done nor is a sphenoidectomy; however, the anterior ethmoid cells deep or lateral to the flap must be well exenterated and the anterior part of the middle turbinate freed and laterally displaced to form a smooth bed for the flaps. Obvious disease is, of course,

removed. When disease is present in the ethmoids and sphenoids on the operated side, these are thoroughly cleaned from above. When these are present on the opposite side also a De Lima transantral ethmoidectomy without frontotomy is done, thus not disturbing the mucosal flap. When disease is present in the antrum, on the operated side a Caldwell-Luc procedure is performed. These procedures can be done either before or after the frontal operation or as a combined procedure.

CLINICAL FINDINGS.

In a series of 21 patients, 20 presented the usual subjective and objective findings of chronic obstructive frontal sinusitis such as *1.* bone destruction, *2.* fistulae, *3.* adjacent orbital signs.

Seventeen of the cases had bone destruction of the floor of the frontal sinus by disease. Three more had a floor defect due to a previous Lynch procedure. One of the original 17 had had a previous Lynch operation but had had floor destruction by disease at that time.

There were three cases with a cutaneous fistula.

Only one of the 21 did not have a bone defect at the time of the mucoperiosteal flap operation.

Fifteen patients had globe displacement. Six of these presented a soft, slightly tender, cystic swelling just above the inner canthus of the eye as in the classic description of mucocele (see Fig. 4). The remaining nine had a previous orbital cellulitis and presented themselves with an edematous, indurated upper lid in various stages of resolution. One of these had left frontal sinusitis which resolved on treatment elsewhere with antibiotics. Two months later he developed blindness in the left eye associated with local pain and swelling of the forehead and upper lid. A diagnosis of optic atrophy secondary to left frontal sinusitis was made when he was first seen in our clinics three weeks later.

There was one incidence of orbital displacement with ophthalmoplegia and pallialedema but normal vision.

Fig. 4. Pre- and postoperative photograph of patient.

In another, a large osteoma involving the floor of both frontals, the lacrimal bone, ethmoid labyrinth and front face of the sphenoid on the right was the cause of nasofrontal duct obstruction and orbital cellulitis.

Of the four patients who had a previous Lynch type operation, one developed a cutaneous fistula, one developed orbital abscess with some globe displacement, and two had recurring swelling of the upper lid which subsided on antibiotics.

Only one of the total number had persistent local discomfort resisting conservative management as the sole indication for surgery.

Bony destruction of the posterior wall of the sinus or the medial wall of the orbit occurred only in the presence of a destroyed frontal floor in this series. In every case, at surgery the nasofrontal duct was found to be obliterated by disease or markedly narrowed.

SUMMARY OF CLINICAL FINDINGS.

Previous Surgery—	
Frontal floor trephine	2
I & D Peri-orbital abscess	4
Lynch type external frontal	4
Intranasal ethmoidectomy	1
Associated Surgery—	
Caldwell-Luc	3
Transantral ethmoidectomy	3
Bilateral Disease	8
Unilateral Disease	13
Etiologic and Pathologic Findings—	
Infection with orbital displacement	9
Mucocele with orbital displacement	6
Allergy	2
Osteoma	1
F. B. Shrapnel	1
Fracture with infection	2
Bone Destruction:	
Floor of frontal—by disease	17
Floor missing previous Lynch	4
Floor missing at first Lynch	1
Posterior wall	6
Orbital Displacement	18
Functional Ocular Symptoms	5
Cutaneous Fistula	3
Total Cases	21

RESULTS.

In this series of 21 patients careful follow-up examination was possible in 20 from one to ten years. One has been lost to follow-up. All but one have been free of any complications. One of the twenty had an episode of mild swelling of one eyelid in the postoperative period. She had had a previous Lynch type external frontal operation which had been followed by recurring orbital swelling soon afterwards, requiring surgery. She had no complications in the later followup. On examining her recently the nasal passage to the right frontal is open but narrow instead of being wide and funnel-shaped at the root of the nose. We think this is the reason for the less than optimal result.

Two have had occasional mild headaches on the side of original pathology. In all cases the new nasofrontal duct has remained open and functioning during upper respiratory infections. During colds their complaints are no more severe than those of other normal people with the same temporary infection.

We feel that in our series we were successfully able to create a new functioning nasofrontal duct in all cases. Boyden[2] has published a much larger series of unilateral flap cases with a longer follow-up, and his results prove that the mucosal flap technique is very satisfactory. We feel that the bilateral flap method permits a much wider opening to be made with less chance of narrowing during upper respiratory infections.

Inactive mucosa lining a frontal sinus, as with mucocele, need not be entirely removed. There are many who would wonder at the advisability of leaving mucocele lining which appears thicker than normal. Actually this lining was left since we have felt a patent new nasofrontal duct is more important than the removal of mucosa that appears thicker than normal. Obviously, we do not advocate leaving intramucosal abscesses or infected lining; however, meticulous excision of all mucosa while theoretically advisable seems to be not necessary at least in our mucocele cases. Return to a "normal" inactive lining apparently occurs.

SUMMARY.

A method of reconstructing the nasofrontal duct is presented. It is based on the work of Boyden and Tato and consists of the rotation upwards and laterally of mucosal flaps fashioned from the lateral wall and septum of the nose. A series of 21 cases is discussed. There were 17 cases with destruction of the bony walls of the frontal sinus, 15 with displacement of the globe and three cases of cutaneous fistula. Mucocele lining has been left without any follow-up complication. The mucosal flap technique was used in all cases and was bilateral in 17 and unilateral in four. In all cases, the method successfully reconstructed a functioning nasofrontal duct.

CONCLUSION.

The common cause of failure in surgery of the frontal sinus is obstruction of the nasofrontal duct. The use of bilateral septoperiosteal mucous membrane flaps from the nose is a very satisfactory method of reconstruction of the nasofrontal duct. Admittedly our series is small, but short term results, from one to ten years, appear satisfactory.

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UNIVERSITY OF TORONTO—POSTGRADUATE COURSE IN OTOLARYNGOLOGY.

On May 11, 12 and 13, 1961, there will be presented a graduate course in this specialty by the staff of the Department of Otolaryngology, assisted by two distinguished guests: Dr. Philip E. Meltzer, Professor of Otolaryngology, Harvard Medical School, Chief of Otolaryngology, Massachusetts Eye and Ear Infirmary; and Dr. W. G. Hemenway, Department of Otolaryngology, University of Chicago.

The first session will begin in the afternoon of May 11th, in the Royal York Hotel, Toronto, in conjunction with the Section of Otolaryngology of the Ontario Medical Association. The remainder of the sessions will be held in the clinical areas of the University of Toronto.

An attempt will be made to assess, discuss and demonstrate the newer procedures employed in the surgery of deafness. The present surgical treatment of head and neck problems will be presented with special consideration of the new conceptions of the responsibilities of our specialty in their management.

The fee for the course will be $40.00 and will include a complimentary dinner.

Please address all inquiries to the Director, Division of Postgraduate Medical Education, University of Toronto.

CLINICAL ASPECTS OF TYMPANIC SOUND CONDUCTION.*

ARTHUR L. JUERS, M.D.,

Louisville, Ky.

Recent advances in the surgical restoration of effective tympanic sound conduction make it necessary that the otologist has a full understanding of the normal, the pathological and the surgically reconstructed sound conduction mechanism. The advent of fenestration surgery provided the initial impetus to understand better the mechanisms of sound transmission to the inner ear. No attempt will be made here to review the complete evolution of this knowledge to date. Particular credit for experimental and theoretical contributions should go to v. Bekesy,[1] Wever and Lawrence.[2] Lempert,[3] Wullstein[4] and Zollner[5] have made contributions on the clinical aspects of the subject. The author has recorded some clinical observations and theoretical considerations, and these previously published ideas will be used together with some additions.[6,7] Many details concerning the mechanics of tympanic sound conduction were presented by Lawrence and Wullstein at the Workshop for Functionally Reconstructive Ear Surgery presented at Northwestern University by Shambaugh in March, 1959.[8]

Four diagrams will be used to show the fundamentals of effective sound transmission to the inner ear under normal, pathological and surgically reconstructed conditions. No attempt is made to conform to normal anatomical relationships. Actually, the wave lengths of audible sound frequencies are much longer in relation to the cochlea than is indicated in these diagrams.

PHYSICS OF SOUND.

Sound can be defined as an audible vibratory disturbance

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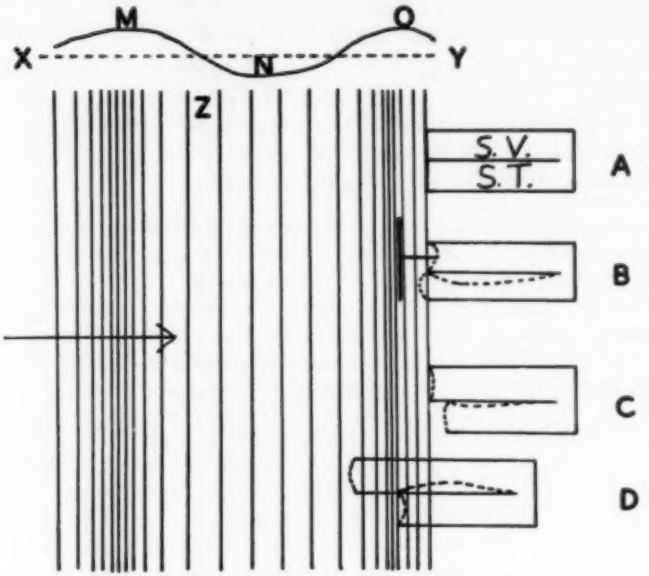

Fig. 1. A.—This is a box representation of the cochlea (v. Bekesy) as applied to the approaching pressure peak. S.V.—scala vestibuli. S.T.—scala tympani. The oval and round windows are at the left and the helicotrema at the right. The basilar membrane with the organ of Corti is indicated by the line between S.V. and S.T. B.—A conduction mechanism placed in contact with the oval window creates pressure differential at the two windows. The pressure is greater at the oval window and the structures move toward the round window as indicated by the dotted lines. C.—The usual anatomical relationship is for the oval window to be closer to the pressure peak than is the round window. Limited movement of the cochlear structures is indicated by the dotted lines. D.—A fraction of a second after C, the pressure peak is at the round window and a reversal of displacement of the structures is indicated.

which can be propagated through any deformable medium. Sound spreads with a circular front in all directions from its source. Sound energy is gradually consumed and dissipated by the inertia of the medium through which it passes, plus the fact that it spreads over an overwhelming area. While sound is usually represented as a curve (see M-O, Fig. 1), it actually is a succession of fronts of positive and negative pressure changes (compression—M, rarefaction—N) with relation to a point of equilibrium. This point of equilibrium is represented in Fig. 1 by the dotted line XY on the curve and at

the point Z in the vertical lines. M-N represents a single vibration of a tuning fork and M-O a double vibration (d.v.).

A full cycle of sound is indicated from M to O, or 360 degrees. The term phase is used to designate a particular point in the cycle. Phase difference is the difference in degrees between two points of the cycle. From M to N would constitute a 180-degree phase difference.

The physical qualities of a medium determine the speed with which sound is propagated through it and also influence the rapidity of absorption of sound energy (decrease in intensity). Sound traverses air at approximately 1100 feet per second, water at 4700 and aluminum at 16,700. This speed differential has some significance in normal tympanic sound conduction as well as in pathological changes.

Alterations in elasticity and/or mass of the conduction mechanism are of particular importance in clinical otology.[9] Any increase in elasticity tends to absorb more energy from the lower than from the higher frequencies. Lesions which fall in this category are early stapes fixation and adhesive limitations of ossicular motion. Since low frequencies of a given intensity normally produce greater amplitude of ossicular motion than do the higher frequencies, it is apparent that a slight limitation of the degree of ossicular excursion will impede sound conduction for low frequencies more than that for higher frequencies.

Fluid in the middle ear with an otherwise normal conduction mechanism tends to cause greater loss for the higher frequencies. The fluid adds mass to the conduction mechanism. As each cycle of sound passes through, the inertia of the total mass—conduction mechanism and fluid—must be overcome twice; consequently, the energy required to overcome this inertia in a given period of time, is greater for the higher than for the lower frequencies.

In A, of Fig. 1, the pressure at the two windows is equal, and there is no movement of the basilar membrane. Bone-conducted sound is not considered here.

In B, of Fig. 1, a conduction mechanism is placed in contact

with the oval window. This accomplishes two things: the wide surface (top of the T) "collects" sound pressure from a wide area and transmits it to the oval window; this collected sound has entered a solid medium, the conduction mechanism, which conducts sound at a much greater speed than is true of the sound which will reach the round window by air. As a result of these two factors, the pressure differential in favor of the oval window moves the cochlear fluids and basilar membrane structures toward the round window as indicated by the dotted lines.

V. Bekesy[10] has shown that the oval window is usually slightly lateral to the round window in relation to the axis of the external auditory meatus. This is indicated in C, Fig. 1. Consequently, even in the complete absence of a tympanic membrane and ossicles, there is some pressure differential at the two windows and some hearing by air conduction. The relative laterality of the two windows is somewhat variable. D, in Fig. 1, shows the reversal of cochlear fluid movement when the pressure peak has passed the oval window and has reached the round window.

NORMAL CONDUCTION MECHANISM.

The following functions can be listed for the normal conduction mechanism:

1. Areal ratio.

2. Round window sound protection.

3. Lever action.

4. Some limitation of excessive excursion in response to loud sounds.

The chief function of the normal conduction mechanism is to provide what is referred to as the areal ratio. The larger surface of the tympanic membrane "collects" sound pressure which is then transmitted to the smaller stapes footplate through the ossicular chain (see Fig. 2). The areal ratio is the ratio of the surface area of tympanic membrane to that of the stapes footplate. The effective, mobile area of the tym-

panic membrane extends between 1 and 2 in Fig. 2. Peripheral to this is the bony rim of the annulus which is noncontributory to collecting sound. The effective areal ratio of the tympanic membrane to the stapes footplate is 14 to one.[8] This adds approximately 30-32 db to the air conduction acuity.

As the sound pressure peak moves the tympanic membrane inward and transmits this movement to the stapes as indicated by the confluence of lines and arrows (see Fig. 2), there is at

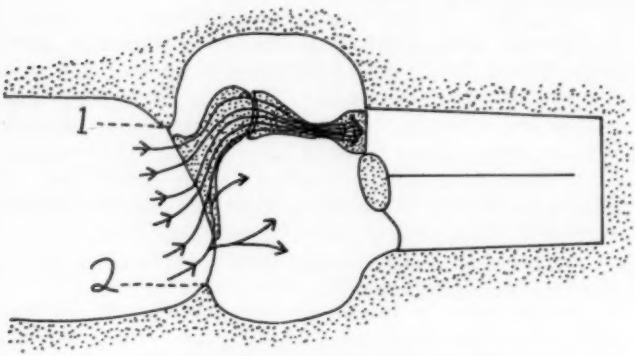

Fig. 2. The mobile portion of the tympanic membrane is between 1 and 2. Sound pressure against this portion of the membrane is transmitted through the ossicular chain as indicated by the lines and arrows. A small amount of sound pressure passes into the air in the tympanic cavity. It is obvious that the pressure transmitted through the stapes greatly exceeds that which passes through air in the tympanic cavity to the round window.

the same time some sound passing into the air of the tympanic cavity. This is dissipated in all directions, going in part to the stapes via air as well as to the round window. In view of this, as well as the fact that the speed of the ossicular conducted sound greatly exceeds that reaching the round window membrane via the air of the tympanic cavity, the round window protection factor of the tympanic membrane is relatively unimportant in a normal ear. This perhaps accounts for the fact that a small tympanic perforation in a normal ear will cause relatively little loss of air conduction acuity.

The lever action of the ossicular chain adds only about 2.5 db to the hearing acuity. The ligaments and muscles at-

tached to the ossicular chain serve chiefly to limit to some extent excessive excursions of the conduction mechanism. This undoubtedly lessens inner ear damage which might otherwise result from some types of loud sound.

PATHOLOGICAL CONDUCTION MECHANISM.

The four chief mechanical changes found in conduction lesions are as follows: *1.* Increase in elasticity, *i.e.*, limitation of excursion. *2.* Increase in mass. *3.* Loss of a portion or all of the tympanic membrane. *4.* Interruption of ossicular chain continuity. These lesions may be present individually or in combination. Certain features of pure tone air conduction audiometric curve configurations are characteristic of some individual types of lesions. Combinations of lesions produce a summation of these characteristics. Variations in the extent of the air-bone gap when comparing low tones and higher tones are sometimes apparent and suggest a certain type of lesion.

Increase in Elasticity.

The most common pathology producing this type of conduction change is either otosclerotic fixation of the stapes or ossicular fixation by adhesive otitis. The earliest shift in the pure tone curve usually shows more loss for the low frequencies than for the high, if there is no impairment of cochlear function. In earlier lesions the air-bone gap tends to be greater for the lower than for the higher frequencies. This could be designated as a converging air-bone gap (going from low to high frequencies). As the fixation becomes complete, the air conduction curve usually becomes more flat. The type of air conduction curve associated with adhesive middle ear changes tends to be somewhat variable but in general shows greater low tone loss in less severe lesions.

Increase in Mass.

Middle ear effusion is the most common cause of this type of conduction loss. The audiometric curve here shows greater loss for the higher frequencies. The air-bone gap tends to diverge when comparing the lower and higher frequencies.

If the effusion is extremely tenacious and completely fills the tympanic cavity and attic area, the curve may be more of a flat type because the nature and extent of the effusion has added an elasticity factor to that of mass. This is the "glue-ear" described by Jordan. A middle ear effusion superimposed on a pre-existing partial ossicular fixation may summate into a flat curve.

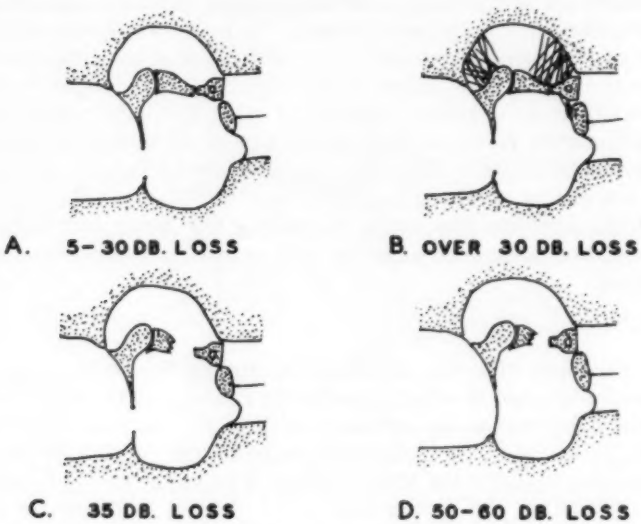

Fig. 3. A.—A central perforation with normal ossicular function and windows. B.—A central perforation with some adhesive impedance of ossicular motion. C.—A central perforation with interruption of ossicular chain continuity. D.—Intact tympanic membrane with interruption of ossicular chain continuity.

The db loss indicated for each of the lesions in these diagrams is the usual average. There are occasional deviations which may be due to anatomical and physical variations in the window areas.

Pars Tensa Perforation.

Fig. 3-A shows a perforation in an ear with a normal ossicular chain and round window. The air conduction loss will vary from no significant shift for small perforations to a maximum of 30 db loss for larger ones. Variable anatomical factors such as relative laterality of the oval and round

windows, overhang of posterior-superior bony meatus over stapes area, axis of external meatus to cochlea could conceivably alter the air conduction threshold.

Fig. 3-B shows some adhesive changes associated with a tympanic perforation. Whenever the air-bone gap exceeds 30-35 db in a perforated ear, the possibility of ossicular impedance or window pathology is likely.

Interruption of Ossicular Continuity.

When there is an interruption of ossicular continuity associated with a sizeable central perforation, the air-bone gap is in the vicinity of 35 db. Variations in window relationships previously mentioned would influence the threshold. If the oval window recess is extremely narrow, *i.e.*, only the capitulum of the stapes is visible between the promontory and the facial nerve, then the sound pressure differential between the two windows would undoubtedly be less favorable.

Interruption of Ossicular Continuity and Intact Tympanic Membrane.

(See Fig. 3-D.) The tympanic membrane here excludes about 15-20 db of sound pressure and the air-bone gap is 50-60 db. This points out the advisability of determining by means of a paper patch placed on the perforation whether closure will improve or further decrease hearing. While interruption of the ossicular chain in the presence of a perforation is uncommon, closure of such a perforation could be embarrassing if such a situation has not been anticipated. Interruption of ossicular continuity in an ear with an intact tympanic membrane is most commonly the result of previous surgical trauma or head trauma. This should be particularly suspected if the air-bone gap is 50-55 db and there is a history of previous mastoid surgery or head trauma.[11]

SURGICALLY RECONSTRUCTED CONDUCTION MECHANISM.

The conduction physiology of a fenestration of the lateral semicircular canal is shown in Fig. 4-A. The tympanic membrane here plays a different primary function as compared to that in a normal ear. The absence of a favorable areal

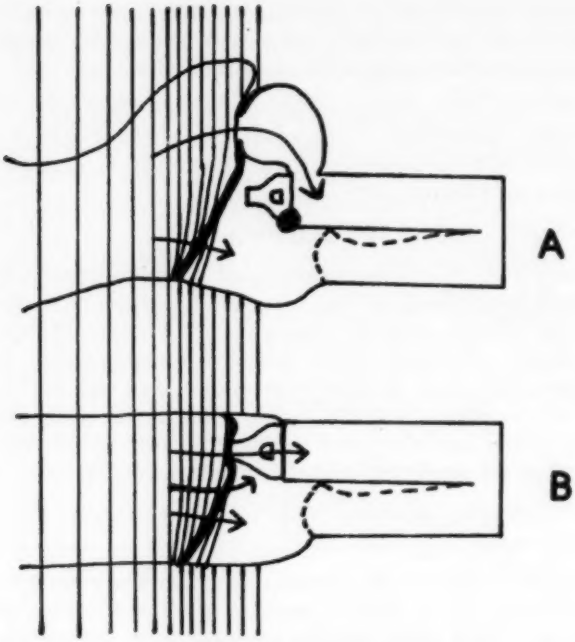

Fig. 4. A.—In a fenestrated ear the pressure peak of a sound wave enters directly through the fenestra in the lateral semicircular canal as indicated by the top arrow. The tympanic membrane serves primarily to "shield" the round window. This increases the pressure differential at the two windows. The stapes is fixed and does not participate in sound conduction. B.—In a myringostapediopexy, the tympanic membrane (or skin graft) is in contact with a mobile stapes. The sound pressure peak is conducted through the stapes. A stapes which is recessed between the promontory and facial nerve would be less favorable to the reconstruction of a good areal ratio than would a stapes which is more exposed.

ratio accounts for the residual "conduction deficit" or air-bone gap of 20-25 db. When the pressure peak approaches the ear, it enters the fenestra first. The tympanic membrane serves to shield or protect the round window somewhat from sound pressure. This increases the pressure differential between the fenestra and the round window. The sound which enters the fenestra travels more rapidly in the fluid of the labyrinth to the basilar membrane than does that which passes through the air of the tympanic cavity toward the round window. If there is fluid in the tympanic cavity, the result-

ing air conduction loss would not be due to a mass effect added to the conduction mechanism. The tympanic effusion in such an ear would rather serve to conduct sound to the round window with greater speed and hence decrease pressure and phase difference between the two windows.

In myringostapediopexy[7] (see Fig. 4-B) the tympanic membrane plays a variable role as far as providing an effective areal ratio is concerned. It is usually technically difficult to arrange the structures to permit establishing a 14-1 areal ratio. This is particularly true in ears which have extensive pathology due to cholesteatoma or chronic infection necessitating a modified radical mastoidectomy. In such cases the sound protection of the round window becomes a more important function of the tympanic membrane than is true in the normal middle ear.

If the conduction lesion is due to an absence of the incus and there is no middle ear or attic disease, then removal of some of the posterior superior bony meatus and the creation of a myringostapediopexy may provide a good areal ratio and closure of the air-bone gap. Shambaugh has described such cases.[11]

An effusion in an ear with a myringostapediopexy would decrease hearing by two possible methods. If a good areal ratio is present, the fluid would exert more of a mass effect. If there was not a good areal ratio, then the fluid would decrease hearing more through the phase difference decrease effect.

SUMMARY.

1. The major function of the normal tympanic conduction mechanism is to provide a favorable areal ratio between the tympanic membrane and the stapes. Lesser functions are to provide sound protection to the round window and a lever action.

2. Early elasticity and mass lesions produce different air conduction curves with converging and diverging air-bone gaps respectively.

3. In a fenestrated ear and in a myringostapediopexy with a low areal ratio, the round window sound protection function of the tympanic membrane assumes greater importance.

4. In an ear with a good areal ratio, thin middle ear effusion decreases hearing chiefly by its mass effect. In an ear with a low areal ratio, middle ear effusion decreases hearing chiefly by its phase difference decreasing effect.

5. In an otherwise normal conduction mechanism, tenacious middle ear effusion filling the entire middle ear will decrease hearing because of its mass effect, elasticity increase, as well as phase difference decreasing effect.

6. Tympanic perforation in an otherwise normal conduction mechanism causes a hearing loss primarily because of a decrease in areal ratio. Loss of round window protection is of secondary importance in this situation.

7. An unusually large air-bone gap in the presence of an intact tympanic membrane is suggestive of ossicular chain interruption, particularly if there is a history of previous mastoid surgery or head trauma.

8. The total displacement of the cochlear structures is dependent on the summation of several factors which influence maximum pressure differences possible at the inner surface of the two windows. These factors include the elasticity and mass of the conduction structures, areal ratio and phase difference increase or decrease effect of structures and media through which sound passes en route to the two windows.

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CRICOTHYROID DISLOCATION.*

JOHN S. KNIGHT, M.D.,

Kansas City, Mo.

This paper was prepared to report the results obtained from the study of nine patients with cricothyroid dislocation.

Review of previous work and literature revealed that dislocations of the cricothyroid joint were first considered to be a common condition but not recognized according to Jackson and Jackson.[1] They state that dislocations of the cricothyroid joint can be traumatic or habitual. The laxity of the ligaments and the habitual manipulation of the patient have something to do with this phenomenon. The first dislocation may occur during yawning or wide opening of the jaws or from external trauma of any form. The displacement is of the cricoid backward relative to the thyroid.

Appearance of the larynx shows nothing abnormal except possibly some feebleness of tension. The symptoms of stiffness, local tenderness and slight hoarseness are present. Thereafter the only symptom complained of was the snapping in and out of place, or the frequent necessity for replacement with the finger.

The treatment does not call for urgent handling. If the patient is constantly annoyed and demands relief, the joints may be invaded surgically to produce ankylosis. Dr. Chevalier Jackson has operated on one case with a successful result. The prognosis indicates that it is not a condition endangering life, and the voice is impaired only slightly. The usual sequel is a habitual dislocation. The patient can dislocate the joint at will, and frequently it dislocates itself to the annoyance of the patient.

The above comments are from foremost authorities, and it

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is not intended to belittle or change their statements; however, experience has shown that the condition as stated above can be classified as subluxation of the cricothyroid joint. In the cases to be presented, a situation has been found that is far more difficult to handle than the mere manipulation of the voice box by the patient. Accordingly, one might presume that a complete dislocation was encountered in a small series of cases, and in two cases an ankylosis also was noted. The dislocation of the inferior cornu has been posterior to the cricoid.

Further study of the literature reveals that dislocations of the cricothyroid articulation have been exceedingly rare, according to Jackson and Coates[2] in 1929. Since that time several articles have appeared in the literature. Kernan and Cracovaner[3] in 1942 described dislocation of cricothyroid joints as rare conditions. Jackson and Jackson,[4] in 1943, described the case of a man who, in an attempt to hang himself, dislocated both arytenoid cartilages and the cricothyroid articulation. Elsbach[5] reported a case of dislocation of the thyroid cartilage in 1948. Gisselsson[6] commented that, judging from the literature, dislocations of the larynx were very rare, indeed. He reported a case in 1950. In 1951, Loeb[7] presented eight patients with cricothyroid subluxation and described such symptoms as pain of the larynx upon swallowing, referred ear pain, and a feeling of a lump in the throat. He noted that there was displacement of the thyroid cartilage either to the right or left. By employing manipulation of the subluxed joint, he was able to effect dramatic relief of pain. Surgical correction was not necessary to obtain a clinical improvement.

The articulation of the lesser horn of the thyroid cartilage with the cricoid is a synovial or true joint. This is essentially necessary for proper and complete approximation of the vocal cords. The thyroid cartilage is movable anteriorly and posteriorly to a mild degree.

Binding the thyroid cartilage to the cricoid anteriorly is the middle cricoid ligament. Surrounding the inferior cornu of the thyroid cartilage with the junction of the cricoid carti-

lage is a thin lax ligament, the cricothyroid capsule. This capsule is assisted or reinforced by the lateral and posterior ceratocricoid ligaments, these acting as a partial check to the movement.

Disturbance of the normal anatomical and physiological relationships of this joint result in certain changes of voice, neck comfort, and ability to swallow.

In the 1945 edition of Jackson and Jackson's[8] "Diseases of the Nose, Throat and Ear" the subject of trauma of the larynx

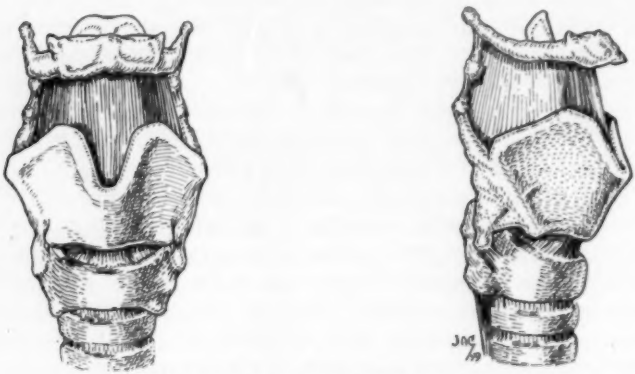

Fig. 1. Normal laryngeal structures.

is discussed quite thoroughly: "Laryngeal injury may be direct when inflicted by an external agent, or indirect when due to action of the laryngeal muscles. In no part of the literature of traumatology is there such a dearth of data as in wounds of the larynx. Most of the cases are buried in the classification of injury of the neck."

Holinger[9] and Proud[10] state that they have not had a recognized case of cricothyroid dislocation in their practice. Lejeune[11] has reported that his experience with cricothyroid dislocations is limited to three or four cases.

Much has been written about the dislocation of one or both arytenoid cartilages, and it is not the purpose of our paper

to discuss this dislocation but to confine our comments to cricothyroid dislocation.

Twenty-nine years ago the author had the privilege of seeing six cases of cricothyroid dislocation of the larynx in a period of one year. Since that time he has not seen or known of any

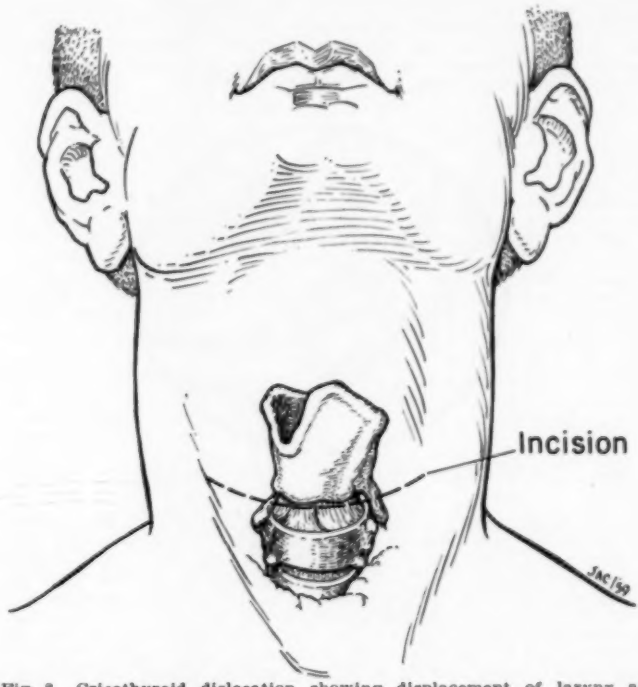

Fig. 2. Cricothyroid dislocation showing displacement of larynx and thyroid notch laterally.

similar cases until the past three years. In this period, two case reports are presented involving cricothyroid dislocation. In each case the dislocation was associated with severe automobile injuries affecting other parts of the body. The dislocations were not diagnosed until four months later in the first case and one month later in the second case. A third

case of dislocation was associated with multiple fractures of the larynx resulting from a blow by a fist.

Case 1. Mr. H.D., a lawyer, age 38, developed his first dislocation in 1929 while leaning back in his chair, turning his head to the side and talking at the same time. He suddenly developed pain and discomfort in the neck and the left side of the face. He came under observation almost immediately. The voice box was rotated to the left, and the pain was referred to the left ear. Efforts at swallowing resulted in a great deal of pain, and there was a huskiness to his voice. The thyroid notch was displaced at least 1 cm. to the left of the midline of his neck. The right side of his voice box was flattened. Indirect laryngoscopy revealed a change of approximately 30 degrees from the midline to the left of the anteroposterior dimensions of his glottis (see Fig. 3).

Fig. 3. Indirect laryngoscopy appearance.

Efforts to reduce the dislocation by manipulation were not successful, and the patient was given an ether anesthetic. It was then very simple to relocate the voice box by manipulation. When the patient awakened he was comfortable. This patient has been seen at various times since his original disability. He says that he is always quite conscious of the act of swallowing, and that he dares not put his head back and talk at the same time. In the course of the past years he has had approximately 20 dislocations of his larynx which he was able to correct himself by inserting his finger into his mouth and pulling upward and forward on the base of the tongue.

Case 2. Mr. J.J.G., age 55, was seen in the same year with the possibility of having swallowed a foreign body in chicken pie. He seemed quite frightened and very uncomfortable. He did not remember pain or discomfort that might have occurred as the result of ingestion of a foreign body. Pain and discomfort particularly on swallowing and with reference to the ear were present. The thyroid notch was displaced to the left side, and indirect laryngoscopy revealed no foreign body but an angulation of the glottis. By manipulation, which included anterior elevation of the base of the tongue and epiglottis with a finger, it was possible to reduce the dislocation with a sudden snap, and the patient immediately became comfortable, stating that something had slipped back, and he felt better. Indirect laryngoscopy showed normal laryngeal find-

ings. There has been no follow-up in this man's case, and we assume that he did not have any further trouble.

Case 3. Mr. F.S., age 60, was an extremely nervous and hypertensive type of individual when first seen in consultation with the complaints of acute discomfort of the throat and referral of pain to his left ear. While he was eating he had turned his head quickly to the left on hearing his name called. The thyroid notch was displaced to the left, and indirect laryngoscopy showed the glottic chink to be angulated 25 to 30 degrees off the midline. He was given a general anesthetic and the dislocation was reduced rather easily. His case was followed for a number of years, and there was no evidence of any recurrence.

Case 4. Dr. T.E., a 25-year-old intern, developed a dislocation with similar findings and complaints that have been described. He was a tall, thin individual. He stated that his first dislocation occurred three years previously after a visit to the dentist. Spontaneous recovery developed in about five minutes. Three or four times a year since then his larynx would dislocate. He learned to relocate his larynx by manipulation. The complaints of sudden onset of pain referred to his ear with difficult, painful swallowing and a lump in the throat were associated with the findings of a displaced thyroid notch and angulation of the glottic chink. By manipulation of the larynx, with particular attention to the maneuver of lifting anteriorly on the inferior cornu, a satisfactory anatomical reduction was accomplished with subsequent remission of symptoms. This patient was last seen in 1931, at which time there was no history of recurrence.

Case 5. R.A., age 13, was choked from behind by a classmate on a school playground and immediately afterward was noted to be quite short of breath, with difficulty in speaking. His mother, a registered nurse, recognized that a potentially dangerous injury to the voice box had been sustained and sought medical attention without delay. On examination, the boy revealed symptoms of referred pain to the right ear aggravated by swallowing, difficulty in swallowing, moderate shortness of breath, and a change in voice with signs of a displaced larynx, and the displacement of the thyroid notch to the right. Direct laryngoscopy revealed a displaced glottic space with angulation to the right, poor mobility of the vocal cords, and submucosal hemorrhage involving the right cord and arytenoid. The glottic space was sufficiently narrowed to warrant an elective tracheotomy under local anesthetic prior to manipulation and realignment of the larynx under general anesthetic. It was considered possible that this patient had suffered a fracture of the thyroid cartilage in addition to the dislocation; however, he made an uneventful recovery and was decannulated within a period of ten days. For approximately six months following surgery some difficulty in developing normal speech was noted, but after this time his voice became normal and there was no discomfort on swallowing.

Case 6. F.D., a 26-year-old, robust, full-necked, athletic type of man, was seen in 1930 with complaints of neck pain and difficulty in swallowing following a sudden turn of the head while sitting at his office desk. He stated, "I felt as though someone had put a knife through my throat but now the pain is behind my Adam's Apple." He did not complain of referred pain to the ear. The voice was slightly husky, there was a feeling of a lump in the throat and an almost complete inability to swallow. There was sufficient shortness of breath to require resting three times during the four-block walk to the physician's office. Examination disclosed a dislocation of the larynx that was successfully reduced by manipulation under general anesthetic, again utilizing the technique of lifting the inferior cornu anteriorly with one hand and elevating the

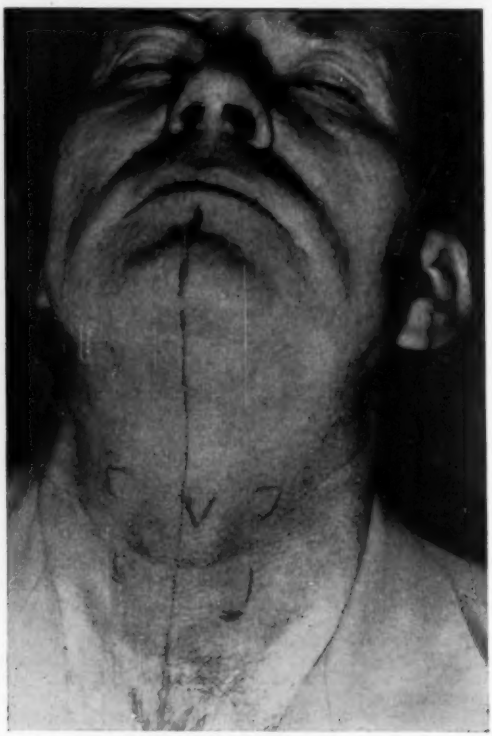

Fig. 4. Case 7, R.B., age 47. Preoperative photograph showing left lateral displacement of the larynx, with "flattening" of the left side of the voice box. This condition had been present for over four months.

base of the tongue with fingers of the other hand. Other than complaints of a sore throat for two days postoperatively his recovery was uneventful.

Case 7. R.B., age 47, was seen on April 16, 1956, on referral because of the complaints of hoarseness, a sensation of a lump in the throat, and discomfort on swallowing which he stated had been present since an automobile accident on January 26, 1956. In the accident his head had been forced through the windshield. He stated that the intensity of the discomfort had been gradually less severe since the accident. Palpation of the neck revealed a displacement of the thyroid notch to the left, and laryngoscopic examination demonstrated an angulation of the glottic space with bilaterally edematous vocal cords and decreased motility of the left vocal cord. X-ray studies showed no abnormalities (see Fig. 4). As attempts to correct the laryngeal deformity by manipulation were unsuccessful it was deemed advisable to utilize an external surgical approach supplemented by simultaneous direct laryngoscopic observation.

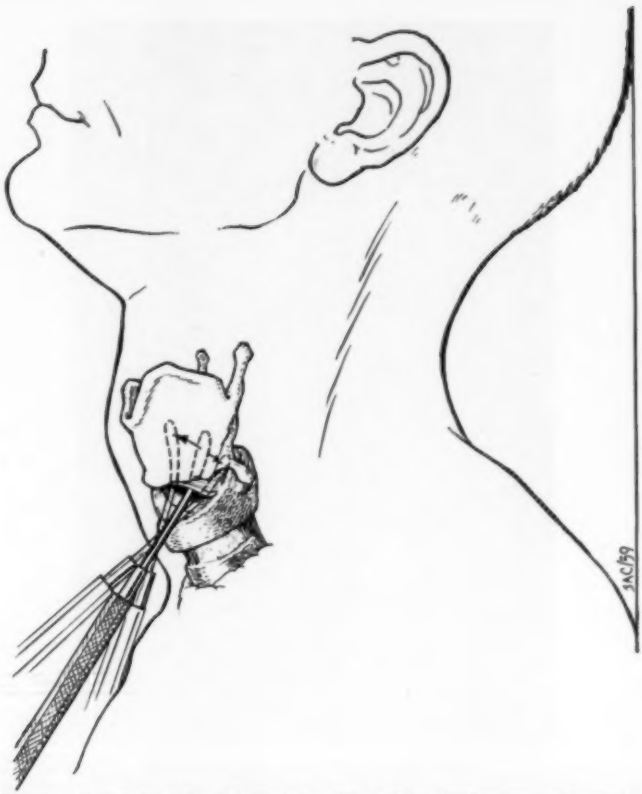

Fig. 5. Surgical approach for relocation with simultaneous elevation of the voice box with the laryngoscope.

Under combined local and general anesthetic an eight-centimeter collar incision was made over the mid-portion of the larynx and the ribbon muscles separated and retracted without disturbing the underlying laryngeal perichondrium. The cricothyroid membrane was cut on each side of the midline being careful not to enter the larynx. A Freer septal elevator was then placed medially to the thyroid cartilage and was used to free the thyroid ala bluntly bilaterally and to reduce the displaced and ankylosed inferior cornu (see Fig. 5). At the time of external manipulation an associate used a Jackson anterior commissurescope to displace the larynx anteriorly; a definite reduction of the deformity was accomplished. The wound was closed and the dressing, fortified by additional packing on the right side, was designed to maintain proper laryngeal alignment. The patient's recovery was uneventful with a marked improvement. When last seen his thyroid notch was in the midline, and he swallowed without discomfort.

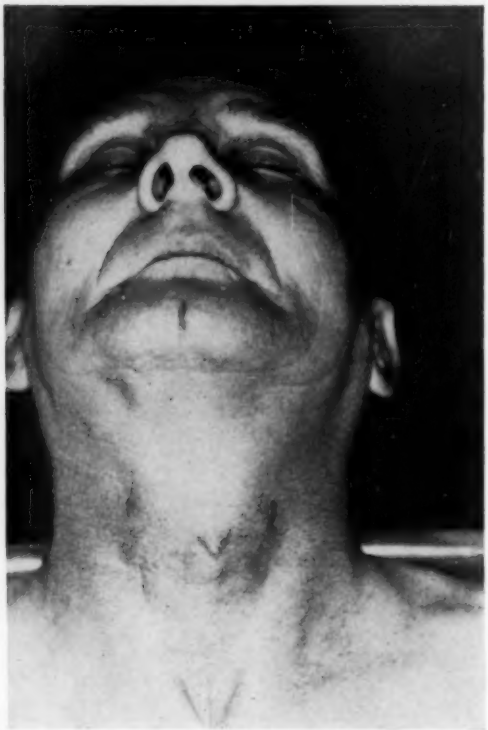

Fig. 6. Case 8, H.B. Preoperative photograph showing displacement. This condition had been present for a month following an automobile injury caused by striking his neck on the steering wheel.

Case 8. H.B., age 47, was first seen on June 12, 1956, a month after sustaining an injury to the larynx and fractures of the legs in an automobile accident. He believed that he had struck his neck on the steering wheel and had experienced marked shortness of breath for approximately one hour after the accident in addition to severe discomfort in the throat and a husky voice. Difficult and painful swallowing with pain referred to the left ear had persisted, and he had found it necessary to remain on a liquid to soft diet. Upon examination it was noted that the thyroid notch was displaced approximately 1 cm. to the left and that there was fullness and tenderness on the right side of the larynx and a flattening on the left side (see Fig. 6). The glottic chink was displaced about 20 degrees to the left in its anteroposterior dimensions. X-ray studies showed no abnormalities. Efforts at reduction of his laryngeal deformity by manipulation were unsuccessful and a laryngoplasty similar to the procedure outlined in the above case was performed. His postoperative

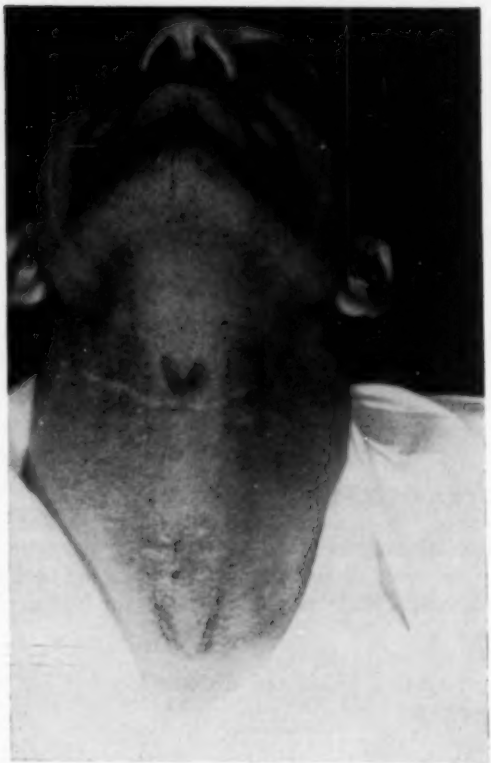

Fig. 7. Case 9, H.C. Postoperative findings of normal external laryngeal landmarks. Laryngoplasty for fracture and dislocation of the larynx two years, 9 months previously.

course was uncomplicated, and there was definite improvement in swallowing function and the location of his voice box. When he was last seen his voice had returned almost completely to normal.

Case 9. M.C., age 49, was first seen in the hospital on July 15, 1956, the day after he had been struck in the neck by one of three assailants. Immediately after the injury he was unable to speak above a whisper and had expectorated a small amount of blood. External examination revealed a swollen, tender larynx with flattening of the left side; laryngoscopy demonstrated angulation of the glottic space to the left with edematous cords and a fractured and partially dislocated arytenoid cartilage (see Fig. 7). Under combined local and general anesthetic, a tracheostomy was performed, and an 8 cm. collar incision was made over the mid-portion of the larynx. After vertical separation and retraction

of the ribbon muscles and evacuation of an overlying hematoma, a fracture of the left thyroid cartilage was evident with five fracture lines visible. The left inferior cornu was dislocated, being displaced posteriorly on the cricoid cartilage. After exposure of the larynx with the Jackson anterior commissurescope it was possible to reduce the dislocated inferior cornu and correct most of the deformity by elevating the larynx anteriorly. A small portion of the exposed arytenoid cartilage was removed through the scope, and the arytenoid was placed in its proper position. A polyethylene tube approximately one centimeter in diameter and five centimeters in length was introduced into the glottis through the scope and transfixed to the neck with nylon sutures brought out laterally and anchored to buttons. The arytenoids and realigned pieces of fractured cartilage were held in their proper positions by the tube and a snug external dressing. The patient made an uneventful recovery. The polyethylene tube was removed 55 days after surgery and decannulation was accomplished one week later. The patient was last seen on April 14, 1959, at which time he was able to speak almost normally, and the outline of the voice box was good. The right vocal cord was noted to have a 20 per cent reduction of motility, the left vocal cord was normal, and the thyroid notch was in the midline.

CONCLUSIONS.

1. Cricothyroid dislocation does occur.

2. Acute dislocations should be reduced under general anesthesia for two reasons: it is more comfortable for the patient, and as in shoulder dislocations, it makes for better relaxation and reductions.

3. Long standing ankylosed dislocations require reduction by open surgery.

4. Subluxation of the cricothyroid joint may be common, but the dislocation is rare.

5. Vague complaints and discomfort in the neck may be the result of subluxation tendency.

6. Early diagnosis simplifies management, and it is probable that the condition fails to be recognized on occasion.

SUMMARY.

Information gained from the study of nine patients with cricothyroid dislocation is presented, and a surgical technique for reducing long-standing dislocations is introduced.

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OBSERVATIONS WITH CERTAIN EXPERIMENTS ON THE PARALYZED VOCAL CORDS IN DOGS.*

MATTHEW H. EVOY, M.D.,

Seattle, Wash.

Injury to one or both recurrent nerves in thyroid surgery is occasionally inevitable, and the consequent cord paralysis is often permanent. The inevitability of nerve damage is acknowledged by anatomists as well as surgeons, as post mortem dissections repeatedly demonstrate the vagaries of the human recurrent nerves. My own findings in cadaveric explorations have shown an absence of a single nerve trunk at mid-thyroid level in fully one-fourth of the cases. These studies, untroubled by haste, lack of exposure, or bleeding, support the findings of Rustad[1] who reported that 20 per cent of human nerves in his series had divided at or below the level of the lower pole of the thyroid.

With a full realization of the inevitability of bilateral abductor cord paralysis on rare occasions, experiments were conducted to simulate as far as possible the actual nerve interruption that may occur in thyroid surgery. With full knowledge that either adductor or abductor components of the nerve can be exclusively injured[2] and thus affect the behavior of the ipsilateral cord, we have attempted to divide all of the recurrent nerve fibers at the thyroid level. With the cord thereby paralyzed, we have studied the possibility of voluntary muscle control of the immobilized arytenoid cartilage by use of the sternothyroid muscle, a muscle that has a voluntary as well as involuntary action.

The movements and actions of the arytenoid cartilages were studied in eight fresh human cadavers that were kindly made available to us by our coroner. In these observations, the

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sliding action at the cricoarytenoid joint was demonstrated. A suture was fixed to the arytenoid cartilage, and, through a window in the thyroid cartilage, the movement of the vocal cord was observed while traction was applied in various directions. With the suture secured just at the insertion of the posterior cricoarytenoid muscle, it was found that the greatest amount of abduction, and hence widening of the glottic chink, was produced when the pull was applied downward, medialward and slightly backward. Such an axis, of course, is exactly that of the acting posterior cricoarytenoid muscle. It was interesting to find, also, that when the traction was released, the cartilage invariably coiled back to bring the vocal cord into the paramediam position. It was our hope that the turgor of the muscles and ligaments about the arytenoid cartilage in the living tissues would also bring about this spontaneous adduction recoil.

METHODS.

Twelve large adult dogs were anesthetized with sodium nembutal intravenously. The vocal cords were examined and then the animals were intubated. A vertical incision was made to extend well above and below the thyroid tubercle. The pretracheal muscles were retracted laterally and the lobe of the thyroid gland identified. The recurrent nerve was almost always visible as a large trunk passing in the trachealesophageal groove. The thin lobe of the thyroid was divided between hemostats and all of the tissue in the space between the carotid sheath and trachea and esophagus was sectioned to insure, as far as possible, the total interruption of the recurrent nerve fibers. In every instance except one, subsequent visualization of the vocal cords established proof of the immobility of the cord.

In each case the immobile arytenoid cartilage was exposed. In four dogs the contralateral sternothyroid was detached from its insertion, brought through a tunnel fashioned between the posterior larynx and the upper esophagus, and the muscle was secured to the immobile arytenoid cartilage. In two animals, fascia was taken from the foreleg and inserted from the contralateral sternothyroid, through a tunnel, and

secured to the articular head of the arytenoid cartilage. In two others, Surgaloy wire was interposed in the same fashion. Segments of autogenous (external jugular) vein were used as a jacket outside of the two fascial struts.

In the last four dogs in this series, the ipsilateral sternothyroid muscle was used. In two instances the muscle was brought directly to the arytenoid cartilage, while in one animal, fascia, and in the other, Surgaloy wire, were interposed.

The animals were reoperated upon in from six weeks to three months following the original procedure, and all were sacrificed after the second operation. The larynx was removed for a more detailed study in three of the dogs.

RESULTS.

At the completion of the initial operation, the cord action was visualized through a laryngoscope while the sternothyroid muscle was activated by electrical stimulation. Almost without exception, some widening of the glottic chink occurred, although the powerful muscle contraction depressed the entire larynx and made observation less than satisfactory.

With the exception of one dog, all animals lost the ability to bark. There was one superficial wound separation, but otherwise the wounds healed promptly without evidence of infection. No signs of inspiratory distress, either resting or exertional, ever developed.

At re-operation, a moderate amount of scar tissue was found in the subcutaneous regions and about the strap muscles on the operated side. The transposed sternothyroid muscle was viable in every instance, and in no case had the muscle or fascia or Surgaloy pulled away from the cartilage. Electrical activation of the sternothyroid depressed the ipsilateral larynx, but in no case was there abduction of the cord as viewed through a window in the trachea. This disturbing and disappointing finding was not surprising in view of the great amount of scar tissue that was found in every animal about the tract occupied by the transplanted muscle, or fascia, or Surgaloy. The articular prominence of the arytenoid carti-

lage was frozen to the adjacent wing of the thyroid cartilage in every case. The cricoarytenoid joint was found to be smooth and free in all instances except one in which a metallic suture was inadvertently passed into the joint cavity.

COMMENT.

It appears that for a few reasons the results of the transposition of muscle in the larynx of the laboratory animal may not be projected to humans. In the first place, voluntary contraction of a somatic muscle cannot, of course, be obtained in animals. Then again, the anatomy of the canine larynx is significantly different from the human, and exposure of the arytenoid cartilage in the dog is quite difficult because of the marked posterior and medial projection of the wings of the thyroid cartilage. The formation of scar tissue, although inevitable, from even the most gentle handling of the tissues, probably was largely a result of mild sepsis in these animals.

Although our observations suggested the futility of successful muscle transplantation about the larynx in the dog, we were encouraged to note that in none of our animals was there evidence that the muscle or fascia or wire had pulled away from its attachment. We had feared that the repeated involuntary elevation of the larynx that occurs with swallowing would place undue tension on the suture lines.

The scar tissue fixation of the arytenoid cartilage to the wing of the thyroid cartilage even where only a Surgaloy strut was employed led us to believe that handling and possibly sepsis were the engendering agents rather than any reaction occasioned by the transplanted tissue. In the two cases in which the fascia was placed in an artificial sheath of autogenous vein, we found no more gliding tendency than in those without a covering. The intima of the vein still glistened somewhat, but was slightly adherent to the fascia throughout the entire length.

CONCLUSIONS.

Experiments with the transplantation of a voluntary muscle to the paralyzed arytenoid cartilage in dogs have been carried

out. It appears that in the laboratory animal, scar tissue fixation of the transplanted muscle, and an invariable fibrous union between the arytenoid cartilage and the ala of the thyroid cartilage, prevent the desired action of the transposed sternothyroid muscle.

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REVERSIBLE SENSORI-NEURAL DEAFNESS.

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Owenton, Ky.,
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THOMAS O. LAWSON, M.A.,
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Frequently the clinician will encounter a patient who shows a perceptive or sensori-neural hearing loss which on later examination is completely or partially reversed. As Juers[6] points out, the assumption that all perceptive deafness is irreversible is no longer valid. Clinical evidence is accumulating which shows that much remains to be learned about reversible, perceptive deafness.

The terminology used in relation to irreversible deafness deserves a rhetorical comment. The ability to send the patient on his way with the explanation that his "ear nerves are dead" or that he has "nerve deafness" is most often a feat of skill. How many diagnostic tests do we have today which pinpoint hearing impairment as specifically related to that short bundle of nerve fibers sometimes referred to as the "hearing nerve?" While the explanation that the nerve is "dead" may serve to satisfy and even impress the patient, the statement is often unsubstantiated and should be relegated to the linguistic junk heap.

Recently the term *sensori-neural hearing loss* has been suggested to replace the historical and much maligned rubric "nerve deafness." The newer term implies that the locus of the loss is cochlear or retro-cochlear without limiting the etiology to the acoustic nerve. A discussion of the published clinical and experimental literature on hearing impairment attributable to the cochlea, central auditory pathway or auditory cortex is beyond the scope of the present paper.

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Juers believes that a broad physiological approach should be applied to the range of etiological factors causing perceptive (sensori-neural) deafness. Certainly a change in our philosophy toward permanent hearing loss is worth considering and should bear much fruit.

Early audiologic evaluation, through otologic and pediatric appraisal and increased research efforts are in order, particularly regarding pre-school children about whom we have the least information.

Improved hearing testing methods beyond those simply determining the degree of loss—objective puretone and speech audiometry, the tonal decay test, the aural overload test, more valid tests for recruitment, diplacusis, tinnitus and the clinical use of delayed speech feedback—offer new insights into the complexity of the auditory mechanism and auditory pathology.

Stapes mobilization and exploratory excursions into the middle ear have added a new chapter to reconstructive ear surgery and to reversible *conductive* deafness. These advances and many others recommend that we adopt a more optimistic outlook toward certain types of perceptive deafness.

Carhart[2] has demonstrated that a portion of the perceptive loss is reversible in successful cases of stapes mobilization. An average increment of approximately 9 db may be expected in the bone conduction thresholds from 500 to 4000 cycles after ossicular function has been maximally improved.

Widespread use of bone conduction audiometry, speech audiometry and tests of cochlear function by audiologists and physicians using standard quantitative audiometric procedures rather than qualitative techniques will permit a more accurate diagnosis of the type of loss and enhance the accuracy of treatment and prognosis.

Miles and Echols[8] reported a case of reversible deafness following surgical extirpation of a pearly tumor of the cerebello-pontine angle. Simonton[13] has presented an excellent discussion of the course of variable deafness in progressive Meniere's disease. There are instances wherein the

hearing returns to normal and then regresses as the nerve cells in the spiral ganglion degenerate. In contrast to the preceding cases, Bordley and Haskins,[1] and Walsh and Goodman[15] call our attention to those patients who manifest an inability to comprehend speech despite a mild-to-moderate puretone air conduction hearing loss. They may have retrocochlear rather than cochlear lesions. Loch[7] cites evidence that a certain percentage of tonal dips at 4,000 cps and 10,000 cps is reversible with proper diagnosis and treatment. The literature is replete with illustrated cases of reversible traumatic deafness.

Mizukoshi, Konishi and Nofamura[9] demonstrated that certain cytochemical changes found in the hair cell nuclei of the organ of Corti are generally reversible. They studied the effects of acoustic stimulation and certain toxic drugs on the metabolism of the cochlea in guinea pigs.

A small percentage of patients show perceptive deafness due to toxic drugs or poisons. Taylor[14] has written an excellent chapter on the effects of drugs and poisons on auditory function. The chapter appears in the classic text, *Medicine of the Ear,* edited by E. P. Fowler, Jr. Sayers and Davenport[11] have presented a thorough review of the physiological effects of CO. The consensus of opinion is that perceptive loss resulting from drugs and poisons is usually reversible, at least in part. Unfortunately, adequate measurements rarely accompany the clinical description.

The authors of this paper encountered a patient who incurred a severe, bilateral perceptive loss as a probable result of salicylates and similar medication administered for rheumatoid arthritis plus an accidental exposure to carbon monoxide. Periodic audiological measurements were made over a 13-month period during the recovery phase of his perceptive deafness. The results are presented in detail and related to several theoretical assumptions.

CASE HISTORY.

Our patient, a white male, aged 42, had been receiving treatment for rheumatoid arthritis. His medication consisted of Pabalate H.C., a salicylate, hydrocortisone compound and chloroquin (Aralen). Complications arose when he was exposed to an unknown dose of carbon

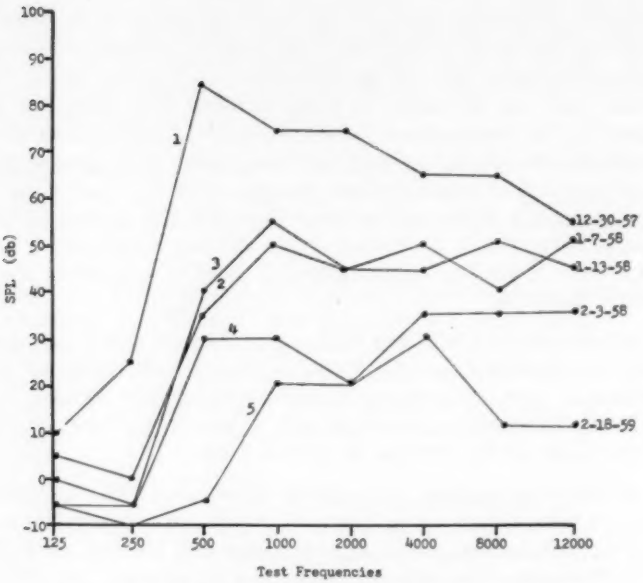

Fig. 1. Right ear recovery (air conduction thresholds).

monoxide from a recently installed, defective damper on his furnace. He was taken to a local hospital for diagnosis and treatment after he was found at his home in a comatose condition. The question of deafness arose due to carbon monoxide poisoning. A week later, December 30, 1957, he was given an audiological evaluation at the University of Kentucky Audiology Clinic.

The results of the audiological evaluation revealed a speech threshold of 80 db in the right ear and a speech threshold of 90 db in the left ear. Speech discrimination was 36 per cent in the right ear and 28 per cent in the left ear. His audiometric loss was verified by Psychogalvanic Skin Response Audiometry, the Lombard Test and Delayed Speech Feedback. Patient complained of tinnitus aurium, bilaterally. He described the tinnitus as "ringing," "chimes" and "hen cackles."

Due to the etiology of his loss, we requested the patient to return a week later for re-evaluation. His retest on January 7, 1958, showed partial recovery of his hearing loss. There was no evidence of diplacusis. He received further evaluations on January 13, 1958; January 23, 1958; February 3, 1958; February 17, 1958; March 4, 1958; March 17, 1958; April 21, 1958, and February 18, 1959.

AIR CONDUCTION THRESHOLDS.

In Fig. 1, the air conduction thresholds for the right ear

are plotted for eight frequencies obtained on five of the examinations. The results show gradual recovery at all frequencies with a residual loss remaining from 1,000 to 12,000 cycles. The thresholds at 125, 250 and 500 cycles are slightly better than normal.

Fig. 2 shows the corresponding thresholds (air conduction)

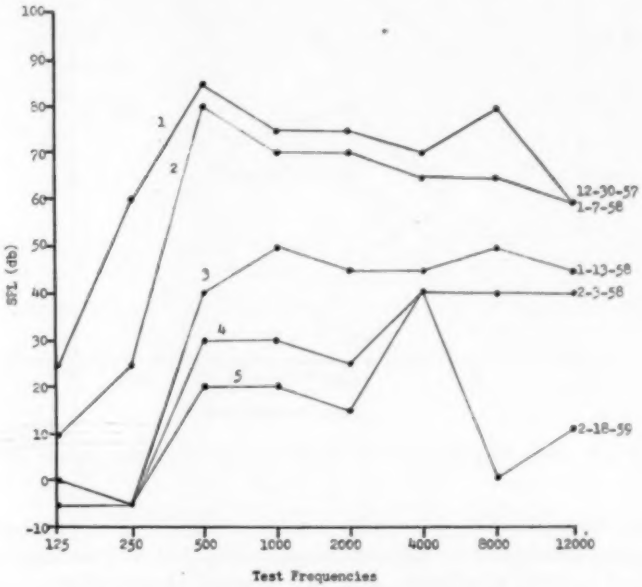

Fig. 2. Left ear recovery (air conduction thresholds).

for the left ear. The recovery is similar to that obtained for the right ear, except that the residual loss extends from 500 to 4,000 cycles with slightly better sensitivity at 8,000 and 12,000 cycles.

BONE CONDUCTION THRESHOLDS.

The bone conduction thresholds displayed in Figs. 3 and 4 parallel the air conduction curves shown in Figs. 1 and 2. On

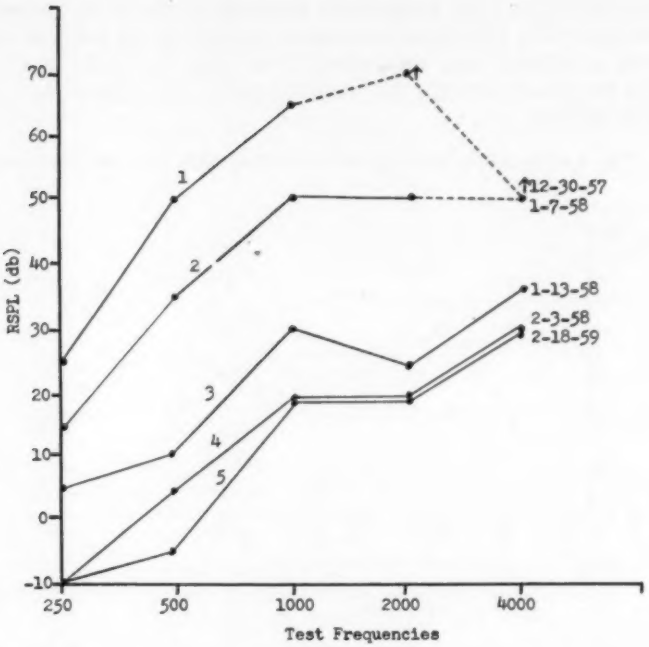

Fig. 3. Bone conduction thresholds during recovery period. Right ear.

this basis, the loss bilaterally would be classified as predominantly perceptive or sensori-neural. The initial examination ruled out the possibility of a psychogenic (non-organic) loss or malingering.

SPEECH RECEPTION TESTS.

The speech thresholds represented by the bar graph in Fig. 5 correlate highly with the average puretone thresholds from 125 to 1,000 cycles. The residual loss for the left ear is reflected in the speech threshold as well as the puretone thresholds. The ten examinations ($t_1 - t_{10}$) permits a longitudinal look at the threshold recovery in the form of the speech thresholds. The right ear recovered gradually from

$t_1 - t_5$. The left ear dropped from 90 db to 20 db from $t_1 - t_6$. The right ear dropped from 80 db to normal from $t_1 - t_5$.

In Fig. 6, the speech discrimination scores for each ear show gradual improvement from $t_1 - t_5$. From $t_5 - t_{10}$, speech discrimination returned to a near-normal level. The reversible nature of this patient's hearing loss is supported by the pure-

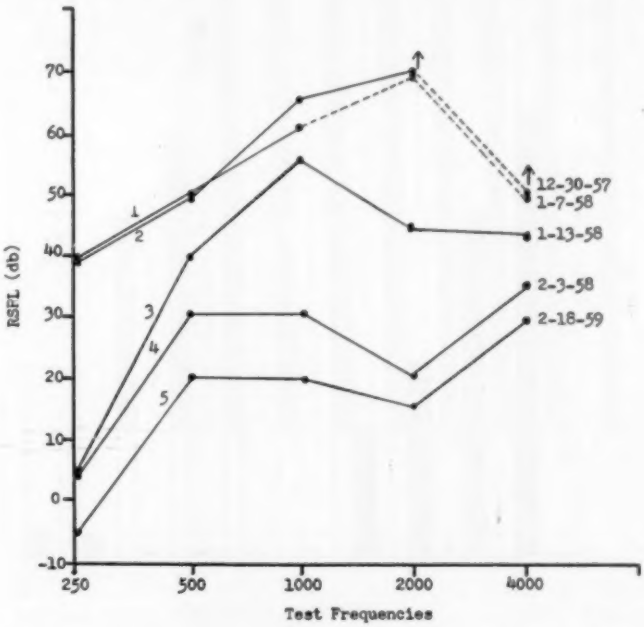

Fig. 4. Bone conduction thresholds during recovery period. Left ear.

tone, speech and bone conduction thresholds and the speech discrimination scores. The patient's subjective evaluation of his hearing always coincided with the gradual improvement demonstrated by the auditory measurements.

On his final examination, there was a diminution in the degree of recruitment and in the loudness of his tinnitus. At

present, he functions adequately in communicative situations. There is a residual puretone loss in the higher frequencies in each ear. Speech discrimination remains near-normal.

Response reliability was consistent during all the psychophysical measures. Threshold fluctuation was not a clinical symptom in this case. No vestibular impairment was noted.

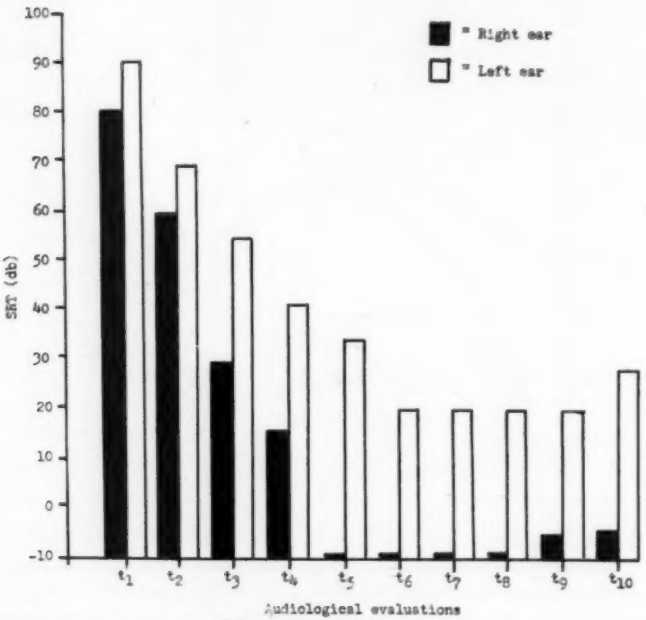

Fig. 5. Speech thresholds measured periodically during recovery period.

There is no evidence of an emotional disorder, and the patient has returned to work. He was also returned to his former medication and dosage schedule.

IMPLICATIONS OF THE DATA.

We are unable to ascertain the separate effects of the salicylate-type medications and the exposure to carbon mo-

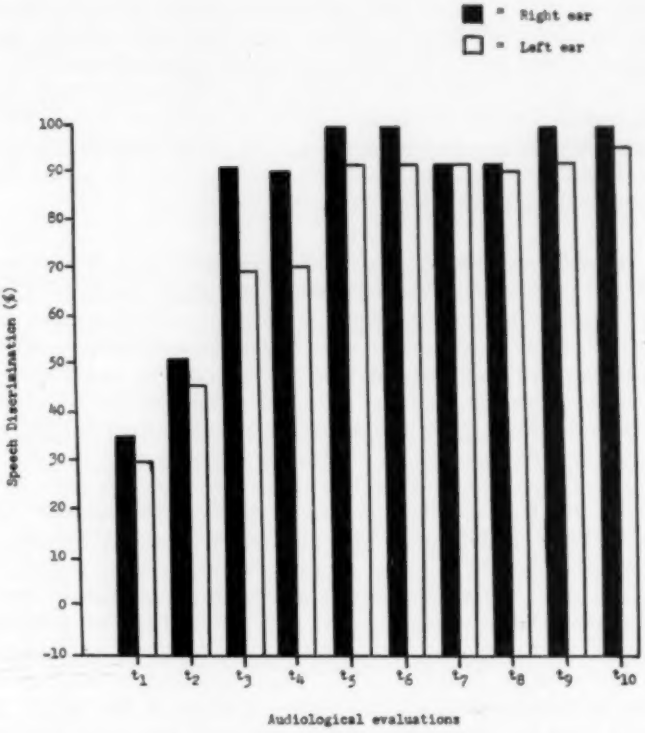

Fig. 6. PB score measured periodically during recovery period.

noxide. It seems worthwhile to relate the aural pathology to certain physiological mechanisms in the auditory system.

The clinical evidence points strongly to a cochlear lesion and a retrocochlear lesion which were at least partially reversible. Ruttin[10] feels that bone conduction impairment and tinnitus are indicative of a cochlear lesion. Recruitment was present bilaterally and is generally considered to have its locus in the cochlea. Diplacusis was not present, even though it is commonly associated with cochlear lesions. Speech discrimination did not decrease with an increase in the sensation level of the stimulus words. In this respect, our case

did not differ from five of the six cases of nerve fiber degeneration reported by Eby and Williams.[5] They also reported that 15 out of 17 cases with end-organ lesions showed a decrease in speech discrimination as sensation level was increased. Their findings appear to support nerve fiber pathology in our patient, since speech discrimination did not decrease when speech was presented at 110 db above the normal threshold.

Dandy,[4] and Schuknecht and Woellner[12] have performed partial resections of the auditory nerve which produced various degrees of hearing loss. The latter experimenters associate diminished speech discrimination with degenerative changes in the spiral ganglion. Crowe, Guild and Polvogt[3] correlated cochlear lesions with high-tone loss in 79 cases. Our patient had a high-tone loss and near-normal thresholds in the low frequencies even after his recovery was stabilized.

The complete clinical picture suggests *both* cochlear and nerve fiber pathology in our illustrated case. We cannot, however, rule out a lesion of the central auditory pathway. A portion of the pathology was reversible since hearing function was largely regained. CO and salicylates may produce changes in the cochlear structures with resultant impairment of the hearing function. The durability and versatility of the auditory mechanism to sustain injury is to be noted. A great deal of pathology may be present with the hearing function showing only a slight loss. This fact may often be overlooked by non-medical specialists. The reversible nature of certain types of perceptive deafness is worthy of critical examination.

The outstanding advances in understanding auditory function and aural pathology offer new hope for the large percentage of the population who suffer from hearing disorders. These advances call for a re-organization of our theoretical considerations of aural rehabilitation. Here is a virgin field for the theoretician, the progressive clinician and the laboratory scientist. Biochemistry and neurophysiology offer audition a major hope for breaching the impaired sound barrier.

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HISTOCYTOSIS INVOLVING THE TEMPORAL BONE.*

(Letterer-Siwe, Hand-Schueller-Christian).

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Thannhauser's textbook (1958) gave the following description of the Hand-Schueller-Christian syndrome: Osseous lesions of eosinophilic xanthomatous granuloma, defects in the membranous bones of the skull, exophthalmos and diabetes insipidus. Greifenstein (1932) treated participation of the hearing organ in this condition in a monograph. After this beginning a well-rounded literature tried to integrate the findings in the ear and in the upper airways into the framework of general knowledge regarding histocytoses.

Fraser (1934) called the clinical entity, in terms of the underlying pathology, lipoid granulomatosis of bones. He felt that endothelial cells from the vessel walls, containing lipoid, pass into the surrounding tissue and provoke lymphocytic infiltration; phagocytic xanthoma or foam cells may contain degenerated leukocytes and even minute bone particles. Arising from the lipoid-containing endothelial cells, their growth results in the multinuclear giant cell.

The monistic theory of Farber (1941) found ever widening acceptance until it reached its present leading role. He considered eosinophilic granuloma not to be a distinct entity but a variant of the basic process of which the clinical pictures known as Hand-Schueller-Christian, and Letterer-Siwe syndromes are other examples; the disease may be suspected on the basis of clinical and roentgenological data, but those have to be correlated with the investigation of biopsy material before a positive diagnosis is established.

Thannhauser explained the concept of the different phases

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as a sequence: Acute reticulo-endotheliosis (Letterer-Siwe) is the earliest, most acute, fatal form; it is followed by a polyphasic phase (Hand-Schueller-Christian), and by the granulomatous and xanthomatous phases. Emerson (1958) formulated the cycle of the manifestations in noncholesteremic conditions as follows: There is an early proliferation of masses of reticuloendothelial cells, including many eosinophils; these masses may be solitary and slow-growing, as in the eosinophilic granuloma of bone, or widespread and rapidly growing throughout the reticulo-endothelial cell system, as in Letterer-Siwe disease, showing a tendency to accumulate intracellular cholesterol and to be transformed into foam cells, typical in Hand-Schueller-Christian disease, and may revert to granulomatous cells with ultimate fibrosis and spontaneous disappearance.

Participation of the hearing organ is mentioned in the very first reports. In Schueller's case (1915), a 16-year-old, otitis media was among the first manifestations of the disease, with defects in the X-ray picture of the temporal squama and pus in the external canal, assumed to be present behind the stenotic canal entrance. Christian's (1920) patient, a five-year-old girl, suffered impairment of hearing among the first symptoms. She heard loud tones, but not the watch; the test was not considered to be accurate as later there was no evidence of deafness.

Reports on manifestations in the ear and in the upper airways were slow to accumulate; but Lederer, Ponchet and Fabricant, in 1935, emphasized the relative frequency of ear-region involvement.

Around the 1950's growing interest and advanced diagnostic methods resulted in rapidly multiplying publications. Some of these showed manifestations in the upper airways to be more frequent than those in the ear. Greifenstein's 26 cases yielded a proportion of 18 to 12; the seven cases of Schuknecht and Perlman (1948) a proportion of four to three. On the other hand, among the 18 cases of Goodhill (1950) the ear participated in 11.

Spontaneous healing or the beneficial effects of X-ray therapy (since 1930) was probably the reason why only biopsy material was offered for histopathologic investigation. Nager stated, in 1933, that in cases of general xanthomatosis a number of clinical reports on the state of the hearing organ were given, but no histological examination of the petrous bone was known up to that date. It took another three years before studies of the latter kind could be added.

Druss in 1936 recorded the ear manifestation of lipoid granulomatosis, Hand-Schueller-Christian disease in a 48-year-old male whose morbid history reached back through nine years with bilateral aural discharge and considerable loss of hearing; in the last seven years he was treated for diabetes insipidus. In numerous bones and soft organs, changes were found corresponding to a universal lipoid granulomatosis; lipoid granulations were found in various parts of the temporal bone but not in the middle ear.

Goodhill registered 18 pertinent cases; 11 had temporal bone lesions, ten with chronic otorrhea. One pair of temporal bones of a three-year-old girl (as reported by Dr. Dorothy Wolff) revealed areas of infiltration into all the pneumatized portions, including mastoid and petrous apex. The infiltrations contained, in various regions, representations of every phase of histocytic granuloma: reticulum cells, foam cells, and eosinophils in clumps. The bony labyrinthine capsule was not invaded. The sensory end organs were intact; the infiltration was restricted to the cancellous bone and the cellular trabeculae of the temporals.

CASE REPORT.

K.T., born September 11, 1955. C.M.C. No. 460061. Referred by Drs. E. F. Carniglia and E. W. Heublein.

This little girl's first symptoms were a seborrhea-like scalp eruption at 11 months of age, increasing irritability by 14 months, and soon thereafter a papular rash on trunk and groin. An exudative eruption was noted on her gingivae, and at 15 months a swelling in the right side of the mandible was shown by biopsy to be an eosinophilic granuloma. There was a temporary shrinkage of this mass after radiotherapy. Severe polydipsia and polyuria began at 20 months of age. An intermittent thin

drainage from the right ear was noted since 17 months of age, but worsened at 21 months and a large swelling appeared behind the ear. At this time, she was first examined at the Children's Medical Center, where it was shown that she had swellings behind the right ear and in the right mandible, accompanied by destructive lesions in the temporal bone and mandible, diabetes insipidus, mild pulmonary infiltration, a scaly scalp eruption, necrotic and exudative stomatitis with loosening of teeth, red papular eruption on chest and back, and mild anemia. Radiotherapy was given to the right side of the skull (including the pituitary area), but the diabetes insipidus did not improve. She was started on repository ACTH (30 units t.i.wk.), which was continued for four months, and chlorambucil (2-4 mgms. q.d.), which was maintained, with occasional interruptions, for the next seven months. Her response to chemotherapy was disappointing, with the skin eruptions continuing in varying degrees, and involvement noted in the perineum and perianal area. Replacement therapy with pitressin tannate in oil was only partially effective, and at 23 months of age another course of radiation was given to the pituitary area. In subsequent months, the impression was obtained that her disease was under some control, in that her pulmonary lesions cleared, new bone lesions did not appear, and the liver and spleen did not enlarge; however, she remained very fussy, and scalp and skin rashes were most troubling. Drainage from the ears, especially the right, was nearly continuous, but it was never possible to establish that this exudate was of any origin other than the moist eruption in the external canals *per se*. The second radiotherapy course had not diminished the severity of her diabetes insipidus, and pitressin injections remained irregularly effective. Prednisolone (up to 50 mgms. q.d.) was substituted for the ACTH (at 25 months of age), but it produced no difference in response.

The final episode began at a time (28 months of age) when her general condition had been relatively good. She developed fever, increased irritability, vomiting, and diarrhea, two weeks after she had had a known exposure to measles (and one week after she had been given a protective dose of gamma globulin). These signs of infection increased, and she required admission to the hospital to aid in the control of fluid balance. There she had her usual skin findings, dehydration, a "toxic" appearance, pharyngitis, and a clear chest to general examination. On intravenous fluid therapy and antibiotics, her vomiting was adequately controlled, but there was a gradual increase in fever, unresponsiveness, pulse rate, and respiratory rate. Finally, she became cyanotic and comatose, and died 36 hours after admission, in spite of extensive supportive measures. Her terminal complications and the virologic studies on her post-mortem tissues are described (as "Case 3") in a recent article by Enders, McCarthy, Mitus and Cheatham (1959).

Autopsy (Pathol. Department of Dr. Sidney Farber) confirmed a Hand-Schueller-Christian process involving the skull, the infundibulum, pituitary stalk, scalp, epicardium, lungs and lymph nodes. In the skull and the infundibular region the process appeared to be in a fairly advanced state of lipidization. Histochemical analysis showed a high proportion of cholesterol inside the lipid-containing cells. From the lungs, affected by a diffuse giant cell pneumonia, measles virus of a high titre was isolated. One of the lymph nodes near the stomach contained Warthin-Finkeldy cells (multinucleated giant cells), while involvement of the lymph nodes by Letterer-Siwe disease was minimal.

Analysis of the tissue lipids (Dr. Allen C. Crocker) yielded the following:

Tissue	Liver	Spleen	Lung	Temporal Bone Tumor
Total lipids—per cent of wet weight	3.91	2.25	2.25	14.4
Phospholipids—				
per cent of wet weight	1.81	1.00	1.13	0.94
(per cent of total lipids)	(46)	(44)	(50)	(7)
Cholesterol—per cent of wet weight	–	–	–	4.39
(per cent of total lipids)				(31)

Analysis of the soft orange mass which had destroyed and occupied much of the right temporal bone showed it to be an extremely fatty lesion, most striking for its cholesterol content—with a phospholipid normal for what would be found in most connective or stromal tissues.

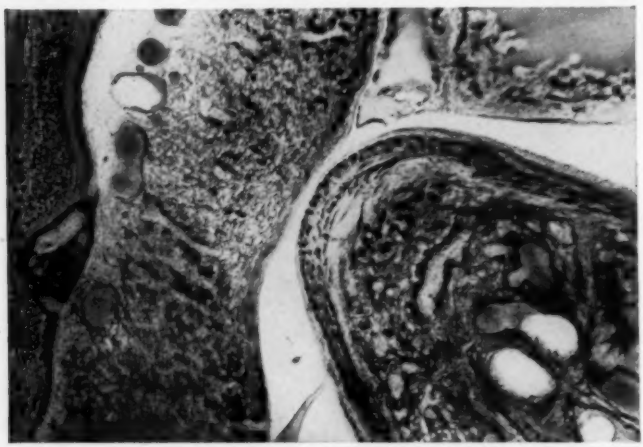

Fig. 1. Gomori, X50. Promontory with Jacobson's nerve; high promontorial mucous membrane; granulomatous blocks suspended in exudate (upper right); tympanic membrane retracted ad maximum without adherence; in external canal, granulomatous mass.

The *right temporal bone* was obtained for detailed examination. Decalcification needed about the usual time; embedding in celloidin, sectioning in the horizontal plane, staining with hematoxylin-eosin, Heidenhain-Mallory, Sudan-Scarlet-red, Gomori impregnation, with unstained sections mounted for polarized light followed.

In the *external meatus* (see Fig. 1) a thick mass completely filled the lumen. Strands of desquamated epithelium were packed together by the granulomatous block which, *in vivo*, probably protruded in form of a polyp. The tympanic perforation, if it was present at all, healed. Retraction of the tympanic membrane brought it into immediate vicinity of the promontory but no adhesion developed.

In the *middle ear* (see Figs. 1 and 2) the osseous walls, except the

medial one, were attacked, resulting in severe destruction. The lamellae of the tympanic tegmen still contained well stained osteocytes; its layers were pried apart by the advancing granulomatous mass; the latter replaced one portion of the bony wall of the hypotympanum. The mucoperiosteum was extremely high (see Fig. 1) even for the young age; some of its very high papillae were branching, and exudate penetrated the interpapillary spaces. High cushions were connected by slender stalks with their bases. In the depth of the mucosa, cysts in rosary formation bore evidence of the chronicity of the process, while engorgement and cellular infiltration marked it to be of inflammatory origin. Cysts were lacking in the niche of the oval window. In the niche of the round window, invasion and organization were present side by side; the

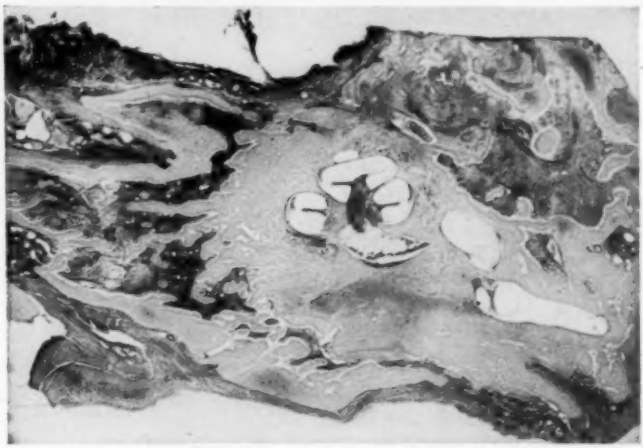

Fig. 2. Gomori, lupe. Survey: center and right lower: intact inner ear; right upper: invaded middle ear with large ossicles, filled by granulomatous mass; left: petrous apex, filled by fibrous mass.

granulomatous mass penetrated from the aperture, while a hyaline but still richly vascularized cushion, in contact with the secondary tympanic membrane, filled the upper medial corner of the niche. The annular ligament was intact and so was the secondary tympanic membrane.

The *ossicles* were completely surrounded by the invading mass (see Fig. 2). The lateral circumference of the capsule of the incudo-malleolar articulation was destroyed, and the articular space was penetrated (see Fig. 2). One corner of the incus was separated as a sequester from the main body. The anterior crus of the stapes was bent in a right angle, near-fractured, possibly by pressure of the invading mass. The latter broke, at the stapes head, through the cortex and filled a considerable portion of the head.

All spaces of the middle ear were filled with an exudate of inflammatory character as shown by clusters of infiltrating cells especially dense in the tubal corner. The granulomatous mass was surrounded by the exudate

and suspended in it (see Figs. 1 and 2). This mass formed a solid wall around the ossicles, filled the niches, surrounded the facial canal. The peritympanic pneumatic cells, like those in the tegmen, were opened by the granuloma, which broke down their septa and filled the lumina.

The invading mass can be described as a granulomatous block with the cells arranged in long rows or sheets, suspended in a dense reticular meshwork and harboring clusters of lymphocytes (see Figs. 3 and 4). Increase of the vascular bed transformed some sections almost into an angioma; the reticular fibers could be shown, by impregnation (see Fig. 5) as arising directly from the vessel walls. Palisades were formed almost exclusively by eosinophils among which, in isolated groups, giant

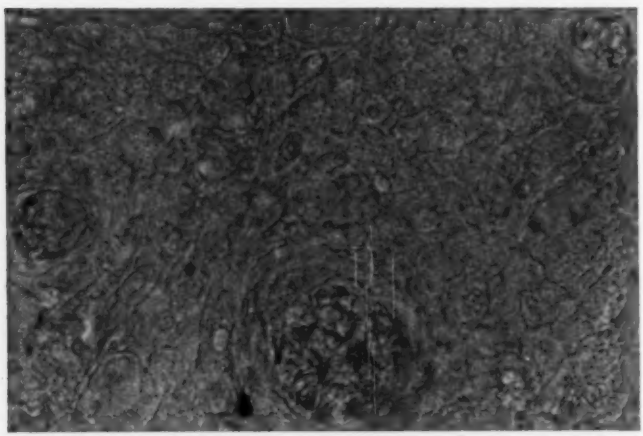

Fig. 3. Sudan, X250. Granulomatous mass in middle ear. Cells in palisades, sheets; field of eosinophil leukocytes and histiocytes; giant cells, partly of foam cell character.

cells were found, and—not too numerous—foam cells. Double refracting elements, sparsely scattered, possibly represented very small bone particles on their way to resorption as a result of destruction.

The *inner ear* remained free of invasion (see Fig. 2). The otic capsule was intact in spite of penetration of the layers of the surrounding portions of the temporal. Destruction started in the operculum, widening the space occupied by the saccus endolymphaticus.

Between the carotid canal and the tip of the cochlea, and sitting athwart the cochlea like a cap, a focus was located of a character which can be best described as angiomatic or spongiotic (see Figs. 6 and 7). It was sharply demarcated against the vicinity, and near the tip penetrated the cochlear capsule, stopping only outside the endosteal layer. It consisted of dense conglomerates of small caliber vessels between the osseous lamellae. The conglomerates did not completely replace the connective tissue of the marrow spaces and did not proceed to form blood lakes by confluence or cause bone destruction. The connective tissue surrounding

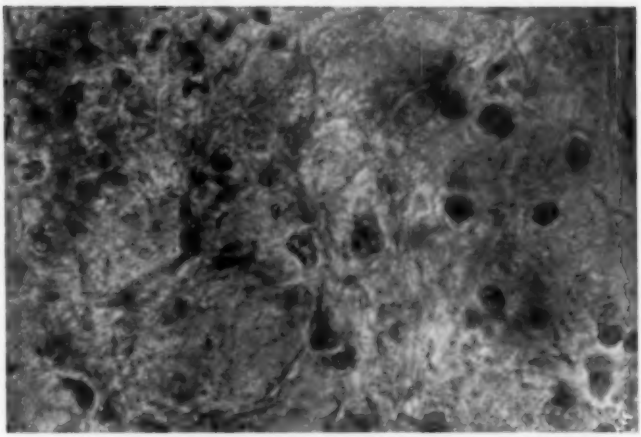

Fig.4. Hematoxylin-eosin, X900. Same granulomatous mass as Fig. 3.

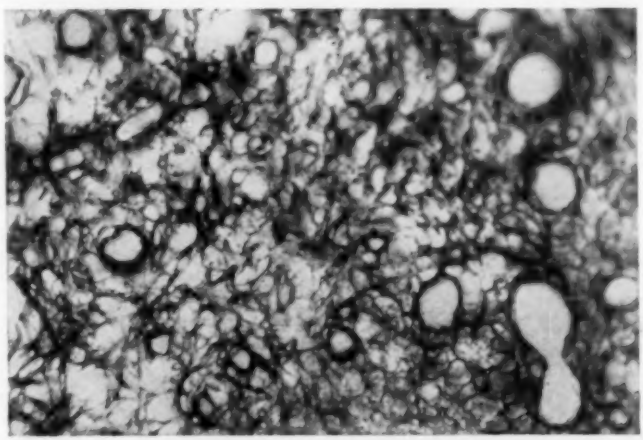

Fig. 5. Gomori, X250. Reticular mesh in the granulomatous mass, the fibers arising from the vessel walls.

Fig. 6. Heidenhain-Mallory, X250. Upper half: Spongiotic focus at tip of the cochlea. Lower half: Normal labyrinthine capsule of the apical coil.

Fig. 7. Unstained, polarized light, X100. Sharp boundary between the spongiotic focus and the normal labyrinthine capsule.

the vessels was best demonstrated by the Sudan stain showing, perhaps, a relation to the basic disease.

The fissular region contained a single large block of cartilage.

The cochlear modiolus was completely filled, the nervous apparatus continuous. The membrane of Reissner was distorted, in places bulging and, again, torn as by overstretching. The tectorial membrane was displaced, without doubt artificially. The organ of Corti (see Fig. 8) was sufficiently preserved to show it to be not reached by the morbid process, and was of normal elevation with its elements well demonstrated inclusive of the hair cells and the hairs. Guttulate formations in the tunnel

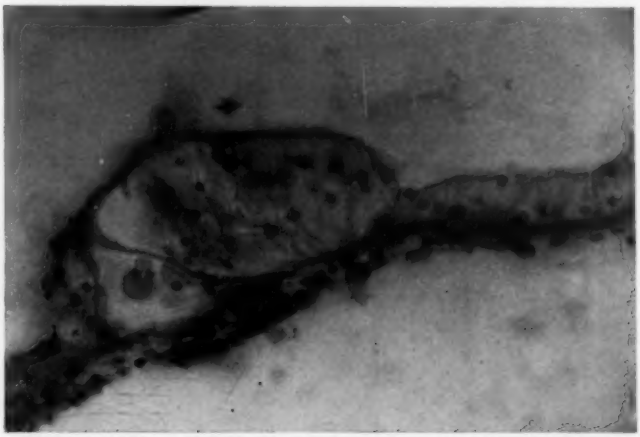

Fig. 8. Hematoxylin-eosin, X400. Organ of Corti in the middle coil. Changes such as rupture of its medial wall, and the guttulate formations, are artificial. Elevation, tunnel, well outlined cells show that in vivo the organ was normal.

and elsewhere within the organ of Corti were doubtless of artificial origin and not stained distinctly by Sudan. The breaking open of the medial wall of the papilla of Corti, with outpouring of cells and loss of the hair of the inner hair cell, has been explained elsewhere (Kelemen, 1956) as originating during the fixation. Nothing abnormal was seen in the vestibular end organs; parts of the vestibular endolymphatic space showed, perhaps, some degree of dilatation.

The inner *acoustic meatus* was free, with a few small psammomas in its dural lining. In its anterior wall a granulomatous focus, part of the penetrating mass, did not reach the surface of the canal wall.

The *petrous apex* was completely invaded (see Fig. 2) but by a very different material from that found in the middle ear. All marrow spaces were tightly filled by a mass made up of fibrocytes and bundles formed by coarser or more delicate fibers. The degree of vascularization was not excessive. The bony septa, still containing well stained osteocytes,

were attacked from every side, with their substance ultimately ground up into small sequesters (see Fig. 9). In spite of the abundant supply of fibrocytes the latter did not line up into rows of osteoblasts; on the other hand, in eroded lacunas, cells were nesting which could be termed osteoclasts by their content of debris. The area of fibrous replacement, going around the otic capsule, reached the base of the high mucous membrane of the middle ear (see Fig. 2).

The walls of the *carotic canal* remained intact; the pericarotic plexus showing engorgement. The *dura* was very thick but without signs of an acute inflammation. The *nervous* apparatus (VIIIth nerve and its ganglia, the facial, the Gasserian ganglion) was not invaded.

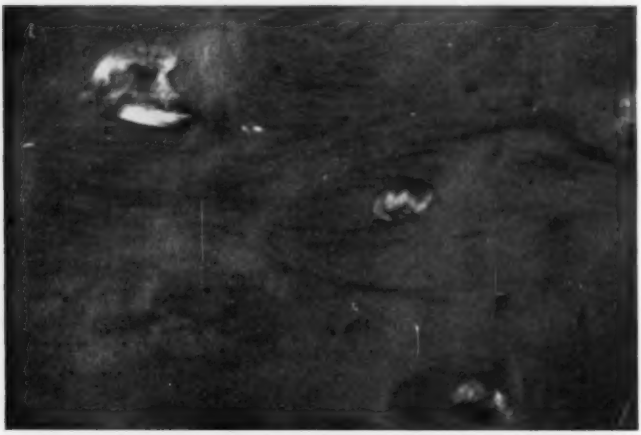

Fig. 9. Unstained, polarized light, X50. Petrous apex filled by fibrous mass, in which smallest sequesters are suspended.

COMMENT.

The foregoing gives histopathological findings in a hearing organ which participated in a generalized lipid granulomatosis involving the scalp, other skin regions, skull, pituitary, both lungs, epicardium and lymph nodes. From the triad (albeit only moderately typical): skull defects, diabetes insipidus and exophthalmos, the latter was absent. Remissions, concomitant otitis media, and measles in the terminal period were recorded. Granulomatous masses filled the external and the middle ear, causing deep destruction of the bone; in the petrous apex complete fibrotic replacement was present, with sequestration

of the septa of the air cells. The otic capsule, with its content, remained intact. Around the cochlear tip a sharply demarcated angiomatic focus was observed in the bone.

The findings must be discussed within the framework of: *a.* the case history; *b.* comparable reports on pathology in the hearing organ; *c.* case reports without complete temporal bone analysis; and *d.* general knowledge on lipid granulomatosis of the bones.

a. In the present case history no exophthalmos was noted. It is known that other constituents of the syndrome, too, may be lacking. Steurer (1952) reported absence of exophthalmos *and* diabetes insipidus. Both, exophthalmos and diabetes insipidus, depend on pressure of the advancing mass, the direction and progression of which are irregular and unpredictable.

Non-involvement of the nervous apparatus of the inner ear depended probably on mechanical protection by the resistant otic capsule.

The role of the measles virus, found in autopsy material, is hard to evaluate; the symptoms were probably covered by the severe basic condition. Measles may have been at the root of the intercurrent otitis media which left signs of chronicity: the rosary of submucous cysts (Kelemen, 1958), the hyalin mass in the niche of the round window, the closed tympanic perforation (in case there was any present at all). Doubt regarding the direct effect of the virus on the sensory structures arose when no pathologic changes as found by Lindsay and Hemenway (1954) could be observed.

b. The case of Druss differed from the present one in the lack of invasion of the middle ear; the latter was traversed merely by fibrous strands, and contained a small amount of purulent exudate. On the other hand the dura was diffusely infiltrated, while here it was thickened, but uninvaded. The two cases exhibited identical features in the osseous parts. Druss described the marrow spaces throughout the temporal bone, inclusive of the petrous apex, as diffusely infiltrated with the lipid granulation tissue, which, to a great extent, underwent fibrotic transformation. Spindle cells containing

lipid substance were scattered throughout this tissue. Contrary to the findings in the present case, there was evidence of new bone formation but no bone resorption around the margins of the marrow spaces.

The case of Goodhill presented infiltrations in all pneumatized portions of the temporal bone, including the mastoid and the petrous apex. No mention was made of any mass in the tympanic cavity; the infiltration was restricted entirely to the cancellous bone structure and cellular trabeculae of the temporal, with areas where either reticulum cells, or foam (xanthoma) cells, or eosinophils predominated.

Filling of the tympanic cavity by granulomatous blocks, suspended in exudate, was seen only in the present case. Druss found a perforated tympanic membrane; through this, parts of the content of the middle ear could have been washed out in the course of the histological preparation.

The most conspicuous finding in all three cases remains the intact otic capsule with all surrounding portions of the temporal invaded. Infiltration of the temporal was described in the two other cases as uniform in character, while here the mastoid and tympanic region were invaded by a cellular-reticular, and the apical region by an entirely different, fibrotic mass. Well stained osteocytes pleaded for a relatively recent date of invasion of the petrous tip, but X-ray showed many months before a "non-osseous" destruction of the pyramid.

The entirely different nature of the invading mass in different locations emphasizes the recommendation of Dawes (1954) and of others to try to obtain adequate biopsy material; a single specimen may not show all the characteristic features of the disease.

To consider fibrosis as a sign of regeneration was here impossible because of lack of new bone formation: the mass of fibrocytes, instead of lining up as osteoblasts, behaved like a tumor, *e.g.*, a fibrosarcoma, destroying the bone by halisteresis.

c. Reports on comparable cases, but without complete histologic analysis of the temporal, contain numerous instances of successful X-ray treatment with arrest or healing up to roentgenological normalcy of the affected bones. Shea (1938) emphasized and was supported in his conclusion by Schall, that surgical intereference should be avoided. Here, the relatively benign course of the Hand-Schueller-Christian phase with multiplicity of clinical and histological features may have been on its way but did not develop sufficiently fast to counteract the acute and fatal Letterer-Siwe phase.

Schuknecht and Perlman found that while the labyrinthine capsule is rather resistant to invasion by carcinoma, this is not the case in Hand-Schueller-Christian lesions. Nager mentioned that participation of the labyrinthine capsule in this disease was assumed by X-ray findings, with a tentative localization in the periosteal layer. Schuknecht and Perlman, discussing differential diagnosis, stated that a very active eosinophil granuloma may produce pictures identical with osteomyelitis of the skull. Steurer's case was diagnosed, preoperatively, as a cholesteatoma. Hertle (1956) reported two cases of frontal bone affection appearing under identical clinical manifestations; one turned out to be osteomyelitis, the other an eosinophilic granuloma. Falkinburg, Littleton, Hunt and Horwitz (1959) published the fourth case with bilateral xanthomatous lesion of the mastoids.

Histologically, here, absence of any reaction in the form of bone apposition discriminated sharply against osteomyelitis with its exuberant osteogenesis amid the most severe inflammatory-suppurative manifestation, right up to the final sequestration (Kelemen, 1957; Meltzer and Kelemen, 1959). Goodhill found the resistance of the capsule of the bony labyrinth to invasion by granuloma remarkable among destruction of other bones of the body, including the ossicles, the annulus tympanicus, the mastoid and the petrous tip. The modiolus of the present case was free of the periostitic reaction to which Brunner (1928) called attention in Gaucher's disease.

According to Dingley (1952) the middle ear is bypassed, at least until a late stage, and non-involvement of facial nerve

and labyrinth may perhaps be so explained. On this basis the present case, with full invasion of the tympanic cavity, is to be classified as an advanced one.

Labyrinth sequesters were repeatedly found. In the case of Chisolm (1954) the middle ear was filled with granulations and a yellowish tissue; the ossicles were absent. On *this* basis—in contrast when comparing with Dingley—the present case, with comparatively intact ossicles, should be classified as a more recent one.

Escher (1950), after operation on a two-year-old girl, found deafness and loss of caloric excitability. This is one example among many of loss of function through the severe process in the temporal.

d. General experience offers roentgenological evidence that the temporal frequently is the only affected region of the skull. If the yellow-gray polyp appears in the external canal, the condition may be diagnosed from the ear alone. Chisolm pointed out that one should become suspicious at any sudden appearance of an aural polyp in a previously normal ear.

Within otolaryngologic discussion, Bartels (1950) considered the single symptom-groups as variants of the same disease. The most outspoken advocate of a "monistic" theory for the entire group of diseases was Goodhill, who defined histiocytic granuloma of the skull as a clinicopathological entity which may appear in an acute, fatal form known as Letterer-Siwe disease, a subacute stage known as Hand-Schueller-Christian disease, and a chronic benign state known as eosinophilic granuloma. He emphasized that the relative frequency of this group should be kept in mind in dubious cases. Dawes was one, and cited others, who accepted this continuity on the basis of experience with cases of ear involvement. The concept of different pertinent lesions as expressions of the same disease was accepted regarding manifestations in different parts of the organism (*e.g.*, McKay, Street, Benirschke and Duncan, 1953).

The present case, considered by the pediatricians as on the borderline between the Letterer-Siwe and the Hand-Schueller-

Christian phase, showed, within the temporal, features which could enroll it under either of the two headings. For this reason it seems justifiable to retain the general term of "eosinophilic" or, as Goodhill suggested, "histocytic" granuloma. "Histocytosis" was adopted here as one of the numerous synonyms offered by Thannhauser.

Regarding *etiology*, Thannhauser asserted that, since an infectious cause was not found, a blastomatous origin of the systemic disorder might be considered. Here, complete lack of osteogenetic reaction in the petrous tip recalled the type of destruction, with simple "melting away," caused by some tumors. There were advocates of the concept to regard Letterer-Siwe lesions as a neoplasm related to leukemia. Actually, as pointed out by Szanto and Saphir (1959), the reticuloendothelial system may react with products either of inflammatory or of neoplastic nature; differentiation between neoplasia and reactive hyperplasia is at times difficult or impossible on the basis of histology. According to Farber the reaction of the bone marrow to the destructive process may be so striking as to suggest a tumor of the blood-forming tissue. It is possible that in the future eosinophilic granuloma, mentioned, as against tumors, as a "tumor-like mass" (Schall and Kelemen, 1959) may attain status as a "true" neoplasm.

The peculiar cap-like, sharply demarcated area in the bone athwart the cochlear tip resembled an angiomatous, spongiotic focus. Cases of otosclerotic changes demonstrated histologically at a similarly early age are known although very rare. The focus found here would represent a spongiotic phase. In this connection it is of interest that Brunner observed Gaucher cells in the marrow spaces of an otosclerotic focus.

In the *therapy*, X-ray, antibiotics, ACTH, and nitrogen mustard compounds were administered in long courses and were considered responsible for the long remissions. The autopsy contained the remark that depletion of the bone marrow in all examined areas could perhaps be ascribed to the long-continued Cortisone. This circumstance is of importance

in explaining the invasion of the pneumatic spaces in the temporal, while the otic capsule, poor in marrow, remained unharmed.

Regarding *prognosis*, the findings in the temporal had shown healing tendency only in the tympanic membrane, rather peculiarly, between granulomatous masses at both sides and this only if a perforation existed, as the discharge may have escaped via progression through the canal walls. This would offer another remarkable instance of higher resistance of soft as against bone tissue. Fibrosis in the petrous failed to act as a cicatrization, and here the mass acted more like a tumor. Had the patient been operated upon, the greater portion of the temporal might have been found—as in numerous reported instances—in the form of a sequester including the still possibly anatomically intact otic capsule.

The short outline of the history of a recently observed case shall be added here in which diabetes and exophthalmos were absent.

A 16-month-old girl was admitted to the Massachusetts Eye and Ear Infirmary (M.G.H. 708722) with a history of an acute episode at the right ear which developed three months previously with fever, anorexia, listlessness, and a profuse discharge from the ear; a polyp was removed but re-formed quickly. She was referred for an operation. At admission the skin was found to be of a yellow (carotinoid) hue; a raised, yellow-brown, papulous area was seen in the left axilla. The left ear was clear. The right canal was bathed in pus, from which coagulase positive staphylococcus aureus and B. proteus were cultured. X-ray showed complete destruction of the entire mastoid process inclusive of the sinus plate and suggested the possibility of Hand-Schueller-Christian's disease. Complete skeletal X-ray revealed no bone lesions. WBC 13,400; hemoglobin 8.6 grams per cent; polys 31 per cent; lymphocytes 61 per cent; monocytes 1 per cent; eosinophils 6 per cent; basophils 1 per cent. She had a moderate anemia; blood cholesterol was within normal limits. Modified radical operation (Dr. Edgar Holmes) revealed a very firm invasive fibrous tissue beneath the mastoid cortex, occupying the mastoid completely and resembling a tumor. The sinus was covered by the fibrous, tumor-like tissue. The posterior canal wall was almost completely destroyed. Polypoid tissue was removed from the epitympanum and the incus was extracted. The dura, exposed over the tegmen of the antrum, has shown no discoloration and no pus under pressure was found under it. The drum did not show any perforation. A skin flap was created from the external canal wall to cover the operative cavity. Specimens of the mass were diagnosed histologically as eosinophilic granuloma. X-ray treatment was inaugurated. Eight years after operation the hearing was normal in the left ear and in the right, air conduction showed for the six tested frequencies, 15, 20, 25, 20, 40 and 30 dbs, with a practically normal bone conduction. At the time of this writing, ten years after operation, the cavity must be cleaned periodically from

massive crust and cerumen, but there is no recurrence, X-ray showing a clean operative cavity.

Several data of this history may serve as a corollary to the findings in the case which is the main object of the present discussion: the temporal, the only involved bone of the cranial base, even of the entire skeleton; lack of two components of the "classic" diagnostic triad; remarkable resistance of the tissue of the tympanic membrane surrounded by the products destructive to the osseous tissue; penetration to the external canal not through a tympanic perforation but by breaking through the bone of the canal wall; re-conditioning of the sound conduction apparatus after eradication of the offensive material; intact nervous apparatus of the inner ear as shown by the good bone conduction ten years after the operation.

SUMMARY.

One temporal bone of a two-year-four-month-old girl with eosinophilic granuloma, who died during the Letterer-Siwe and the Hand-Schueller-Christian phases, was examined in serial sections and compared with two others, the only cases previously reported in the literature.

The tympanic cavity was filled with exudate and residuum from a concomitant otitis media, with the granulomatous products suspended in these secretions.

The granulomatous mass spread to every section of the tympanic cavity and filled the external canal; it penetrated the periantral and peritympanic cells after destroying their septa. Reticular cells, eosinophils in strands and sheets, scattered giant cells and, in distinct groups, foam cells constituted the mass, all suspended in a reticular mesh. Near-angiomatous vessel groups formed at many points. The petrous tip was completely filled by a fibrous mass, very different from the content of the tympanic cavity; it contained, between the fibers, dense masses of fibrocytes which, however, did not line up to form osteoblastic seams. Signs of new bone formation were absent. The otic capsule and its contents were intact, as conforms with the two other published cases. A well demarcated angiomatic or spongiotic focus in the capsular bone surrounded the apex of the cochlea like a cap.

The histopathological findings supported the monistic concept according to which the single groups of symptoms in this sequence of diseases represent only different phases of the identical basic affection.

ACKNOWLEDGMENT.

Thanks are due to Allen C. Crocker, M.D., who contributed the condensed history, the data of the analysis of the tissue lipids, and who helped to set the special findings into the framework of the general condition.

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DISTINGUISHED SERVICE AWARD GIVEN TO NEARLY DEAF EAR PHYSICIAN.

Dr. Kenneth M. Day of Pittsburgh, Pa., is recognized by his medical colleagues as one of the most successful ear physicians in the country.

He attributes his success to the fact that he has been nearly deaf himself for many years, and has served as a model of what the hearing handicapped can really do.

In honor of his service to medicine and mankind, Dr. Day received a distinguished service award May 20, 1960, from the Pennsylvania and New Jersey Academies of Ophthalmology and Otolaryngology at a joint annual meeting in Atlantic City, N. J., attended by eye, ear, nose and throat physicians from six middle Atlantic states.

THE FALSETTO. A HIGH SPEED CINEMATOGRAPHIC STUDY.*†‡§

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MAURICE LE COVER.

Centuries ago the old Italian singing masters recognized two registers of the human voice, the chest and the falsetto or head, the latter two being synonymous. With the invention of the laryngeal mirror it became possible to supplement acoustical impressions with direct observation, and individuals of varying degrees of knowledge, training, and skill recorded their findings and interpretations. The effect of this was to throw the matter of registers into almost hopeless confusion by the introduction of new and often contradictory concepts and terms.

Within the past 100 years the falsetto has been described by vocal pedagogues as a definite register identical with the head voice, as a register distinct from the head voice and situated above it, and then again as below it, and as not being part of the singing voice at all. Since the term falsetto conjures up a variety of musical images, it is imperative that any discussion of the falsetto be prefaced by the author's explanation of what it connotes to him, so that one major source of confusion can at once be eliminated.

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§A sound motion picture illustrating these studies may be obtained from the senior author upon request.

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Fundamental changes in the mechanics of phonation occur at points throughout the singer's range. These are manifest either by an audible "break" or "crack" in the relatively untrained singer, or by a less violent shift in the more experienced singer, as he attempts to accommodate to the physiological and/or the acoustical demands of the new pitch.

One such production change is most apparent as the voice enters its upper reaches and assumes a less complex and a lighter quality. For the purposes of this discussion the falsetto is defined as *that portion of the voice which encompasses this upper singing range.*

The vocal cords in falsetto vibrate so rapidly that the actual nature of their movements can only be conjectured on indirect laryngoscopy; nonetheless, observers in the last century, handicapped as they were by relatively primitive methods of visualization, agreed on two aspects of the falsetto which more refined methods of examination have proven to be substantially correct: first, that in the falsetto a smaller amount of thyroarytenoid substance is thrown into vibration; second, that in the falsetto, the vocal cords are activated by a less powerful air blast. Any further considerations prior to the era of high-speed photography soon bog down in a morass of controversy, and the interested reader is referred to excellent presentations by Mackenzie[1] and Negus.[2]

It was not until the Bell Telephone Laboratories[3] investigated the mechanism of the larynx by high speed photography that real objectivity was possible. According to the brochure accompanying their motion picture the films show that in the lower register the thyroarytenoid muscles strike against each other in phonation with varying force whereas in the falsetto only the margins vibrate, and complete closure is usually not attained. It is also inferred from this study that there is no significant difference between the production of the falsetto and that of a low intensity sound.

Superb as these high-speed motion pictures are, their scope in the study of the falsetto is limited since camera speed did not exceed 4,000 frames per second, and only untrained voices were studied. In the higher reaches of the falsetto, particu-

larly the female, a speed of 4,000 frames per second is not enough to allow detailed analysis; furthermore, such a study of the singing voice would seem to require subjects with trained as well as untrained voices, not only for comparative analysis but also in order to assure controlled performances in the fullest possible range and dynamics of the pure falsetto.

This report will evaluate conclusions drawn from the films of the Bell Telephone Laboratories in the light of additional high-speed photographic evidence. It will also clarify other features of the falsetto which heretofore have not been demonstrated. These include *1.* the mechanism of the so-called falsetto "break"; *2.* the nature of vocal cord activity within the falsetto itself during changes in pitch and intensity; *3.* the means by which the trained singer effects smooth transition from the fundamental register to the falsetto.

METHOD OF INVESTIGATION.

Motion pictures of the vocal cords of selected vocalists were taken by indirect laryngoscopy at 6,000-8,000 frames per second with a Wollensak Fastax camera. The technique, which is the subject of a separate communication,[4] constitutes a radical departure from that devised by the Bell Telephone Laboratories in that the laryngeal mirror is freely manipulable rather than fixed. The elusive falsetto "break" was captured by utilizing film in 400 rather than the usual 100-foot rolls. Since this allows three-and-one-half to four seconds instead of only one second in which to photograph a phonatory sequence, it was possible to encompass the "break" or any other isolated phenomenon. Voices were recorded simultaneously by a microphone applied to the neck over the thyroid cartilage.

The subjects were voice students in varying degrees of training from the department of music at the University of Southern California and professional singers of the authors' acquaintance. The vocalists were required to produce a legitimate falsetto, possess a larynx capable of easy visualization, and be able to tolerate a laryngeal mirror. These criteria were enough to exclude many otherwise vocally qualified

subjects. Of those screened, 15 men and 17 women proved acceptable, and their efforts form the basis of this report.

FINDINGS.

The Falsetto "Break."

One of the aspects of the voice which has eluded analysis is the so-called falsetto "break," a term applied to the sudden change in quality of the voice as it passes from the lower register to the falsetto and vice versa. A prime object of vocal training is the covering of this "break" so that a smooth transition may be effected. High-speed films at the moment of transition reveal a sequence which is constant in both sexes at varying levels of intensity and the same whether the "break" occurs on a glissando or on one pitch. Variations in appearance are related only to the degree of vocal skill.

In ascent or in transition on the same pitch (see Fig. 1) the vocal cords are first seen vibrating in the manner characteristic of the lower register, striking each other regularly along their full length and participating with their entire mass in vibratory activity, but very taut because the pitch is high. As the "break" is reached, the movements become irregular and without any discernible pattern, the vocal folds wobbling loosely without touching each other, resembling banners waving in a breeze. The picture is one of complete chaos. The vocal cords of the untrained vocalist may actually come to a halt as the air stream momentarily stops while the neophyte strains to negotiate the "break." In the trained vocalist this period of lack of control is short-lived, and the manner in which the "break" is avoided altogether in the glissando will be discussed later.

After a varying period of irregular movement, a quite different pattern of vibration emerges. The vocal cords again move in phase and regularly but with a changed overall appearance. They vibrate at the same or approximately the same rate but touch each other lightly or not at all; moreover, their margins are quite thin, and the main mass of the thyroarytenoid muscles lies laterally and does not appear to be

Fig. 1. Falsetto "break." At the moment of transition from fundamental to falsetto or visa versa, the vocal cords are not under control and vibrate without any definite pattern.

participating in the vibratory activity. This is the picture of pure falsetto. The same occurs in reverse as the voice passes from falsetto to lower register.

What transpires in this sequence is easily deduced. In the uppermost reaches of the fundamental register the vocal cords are in a state of maximal tension. Opposing them are the antagonistic cricothyroids which have approximated their points of origin and insertion almost to the utmost. Tension is so great that further elevation of pitch by extension of this

same mechanism is impossible, and a quick muscular readjustment takes place. The thyroarytenoids relax and in so doing are blown about desultorily. The glottic chink in the poorly trained vocalist is prevented from being elongated precipitously at this moment from unopposed cricothyroid pull, because the latter muscles are already in a state of almost maximal

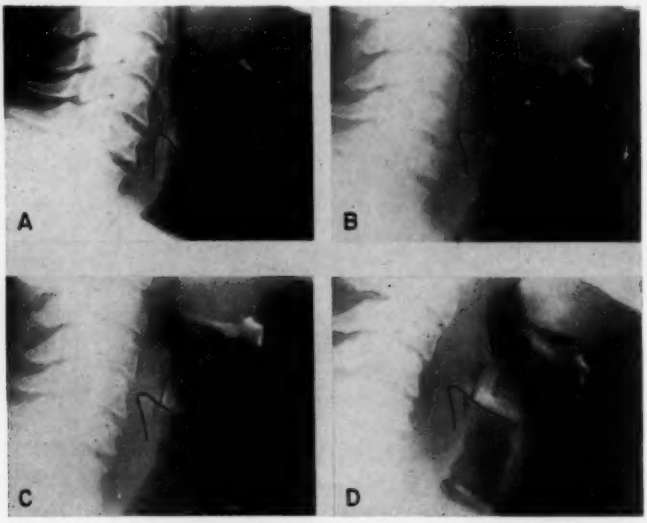

Fig. 2. Lateral X-ray of the larynx in an untrained singer. Maximal elongation of the glottic chink occurs in passing from low to high fundamental registration; very little in the falsetto. Legend: A.—Low fundamental; B.—High fundamental; C.—Low falsetto; D.—High falsetto.

contraction from having previously counteracted the thyroarytenoids.

After a variable period of uncoordinated or poorly coordinated movements, the pure falsetto vibratory pattern appears. The main mass of the thyroarytenoids now lies laterally and no longer vibrates. It would be incorrect to assume that they are completely relaxed because if this were true, they would continue to be blown about as in the "break" or as is often seen at the conclusion of phonation. They are rather in a

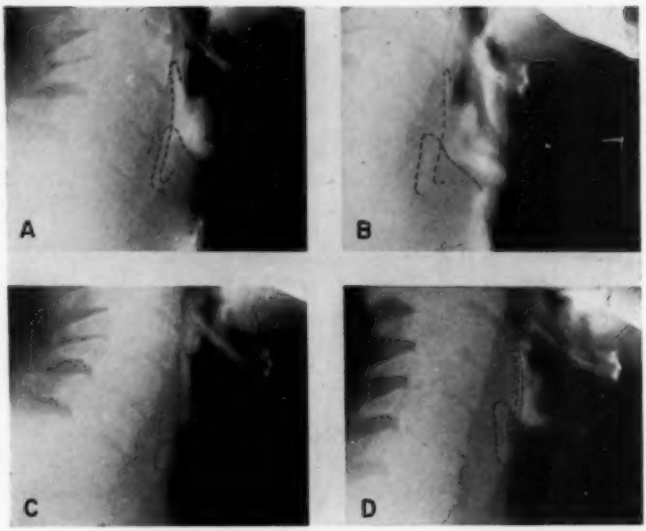

Fig. 3. Lateral X-rays of the larynx in a trained singer. The glottic chink elongates in passing from low to high fundamental registration, not at all in the falsetto "break," and little or none in the falsetto. Legend: A.—Low fundamental; B.—High fundamental; C.—Low falsetto; D.—High falsetto.

state of tonic contraction sufficient to withstand the air-stream yet not enough to bring them together or at best only lightly in the midline. Only the margins now vibrate. This presents a thin, poorly supported resistance to the air column and, as a result the cordal edges are forcibly separated in each vibratory excursion before they are able to make any or more than light or partial contact with each other.

Lateral X-rays of the neck in low fundamental, high fundamental, low falsetto, and high falsetto reveal that there is considerable elongation of the glottic chink in passing from low to high fundamental. There is, however, no further lengthening in transition to falsetto and little or none within the falsetto itself (see Figs. 2 and 3).

TYPES OF FALSETTO ACTION.

Some vocalists presumably have no falsetto, and one is

elicited with difficulty, if at all. Others have a falsetto but never use it or do so only occasionally and for special effects. Still others have a wide range and are capable of singing in the falsetto with exquisite delicacy and beauty.

The physical basis of the falsetto is revealed by high-speed photography to be an astonishingly complex series of intralaryngeal muscular adjustments. Common to all of them and distinguishing the falsetto from the fundamental register is the lesser amount of thyroarytenoid substance thrown into vibration by the air column. By maintaining a high degree of tension in the medial marginal fibers of the thyroarytenoids and by relative relaxation of the main muscular mass so as to avoid its directly confronting the air blast, the higher pitch and thinner quality of the falsetto are produced. There are several ways in which this is accomplished, and, despite much overlapping, three basic patterns of vibratory activity can be identified. One or two or all three may be present in any one voice.

The first of these has already been described in connection with the falsetto break (see Fig. 1), and is characterized by vibrations in which the vocal cords touch each other infrequently or not at all. This "open-chink" mechanism can never be confused with any part of the fundamental register and to see it in a high-speed sequence is to know that the subject must be singing falsetto. Since the main mass of the thyroarytenoid muscles is selectively relaxed, only the thin cordal margins are thrown into vibration by the lessened air pressure from below. This particular mechanism is complete within itself, and the entire falsetto range may be encompassed by it at soft and moderate intensities. Sometimes noted in this action is an anterior to posterior wave-like rolling or "rocking horse" movement of the vocal cords in which, within the same vibratory phase, the posterior halves of the vocal cords are separating as the anterior are coming together.

The second and most common of the vibratory patterns in those voices studied is one in which the margins of the vocal cords strike each other as in the fundamental register (see Fig. 4); however, the main mass of the thyroarytenoids

Fig. 4. Closed-chink falsetto mechanism. The vocal cords touch each other in vibration, but there is relative inactivity of their lateral portions.

is relatively less active, principle participation being by the medial fibers.

In well-trained voices, particularly the female, because of superb control of the thyroarytenoids, there is sometimes difficulty in differentiating this "closed-chink" action from the upper levels of the lower register; and only the tape recorder will suggest how the tones were produced. Even then it may be impossible because the skill of some vocalists is so great that the listener is deceived and cannot at all be certain of

the registration. For the most part, however, they can be distinguished on film.

Actual measurement of the glottic wave vibrations by high-speed photography in both head and chest registration were recorded by Timcke, et al.,[5] and vocal cord movements within each vibratory cycle described in terms of an Open Quotient and a Speed Quotient. These formulas are a real contribution in providing a framework for objective analysis of high-speed film sequences. A detailed consideration of our films by this method, which requires careful attention to specific pitch and intensity, will be the subject of a separate communication.

Suffice it to say here that the vocal cords in the "closed-chink" type of falsetto action appear during projection at usual running speed to be in contact with each other for a shorter period than at comparable intensities in fundamental registration, and they give the illusion of springing apart more quickly after contact. It is as though in the resting state the vocal cords would lie together in fundamental and apart in falsetto.

In fortissimo the usual falsetto action is closed-chink, the vocal folds being pushed laterally to such an extent by the blast that they bounce together on the rebound. A certain number of these will revert to open-chink when the intensity lessens.

A delicate balance may be achieved between the open-chink and closed-chink types of falsetto action, which are really only degrees of the same basic mechanism, the two alternating with each other in producing a single sound. This represents the ultimate in balance between tension of the cordal margins and subglottic air pressure. In this situation the air stream forcibly separates the apposed vocal folds, but the frequency of vibration is so high that the margins spring back toward each other before enough air has escaped to allow them to come together. They are thus held apart until subglottic air pressure again reaches the point where they are blown apart. Since they were not approximated tightly in this phase, less

air pressure is required to push them laterally, and a greater drop in air pressure occurring at this instant allows the vocal cords to make contact again. The cycle is repeated at high frequency down through the falsetto.

In a number of singers, particularly females, a phenomenon was noted in high falsetto and occasionally in high funda-

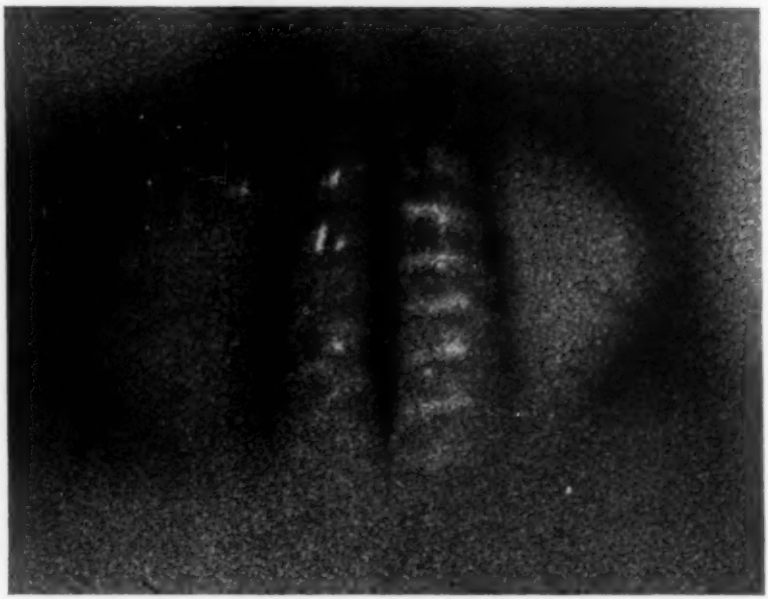

Fig. 5. Ridging in a female vocalist singing high C. At high frequencies mucus not usually seen on the superior surfaces of the vocal cords is sometimes aligned in visible parallel strands which reflect the light and cause a "stepladder" appearance.

mental registration which has not been previously described. This is horizontal ridging on the upper surfaces of both vocal cords, the light reflexes at times being so pronounced (see Fig. 5) as to resemble a stepladder. Frequently associated with this are irregularities of the cordal margins (see Fig. 6) giving rise to a scalloped appearance during vibration. This

was at first thought to represent an unusual type of configuration occurring at high frequencies, but close study revealed it to be due to mucus. In some subjects, at very high rates of vibration, the thin layer of mucus on the superior surfaces of the vocal folds aligns itself in parallel strands which reflect the light. As the cordal margins spring apart during rapid

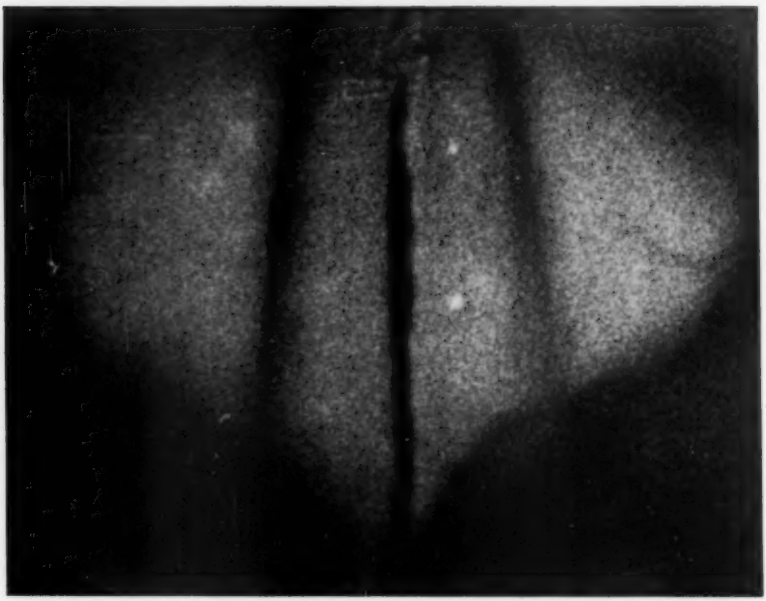

Fig. 6. Scalloping in a female vocalist singing high C. At high frequencies mucus not visible at lower rates of vibration sometimes produces an irregular appearance of the vocal margins.

vibration, this mucus is left behind momentarily because of its inertia, thereby giving them a somewhat irregular or sawtooth shape.

The third type of falsetto action is stop-closure or damping. This becomes operative in the highest reaches of the falsetto in some voices and allows the pitch to rise beyond that possible

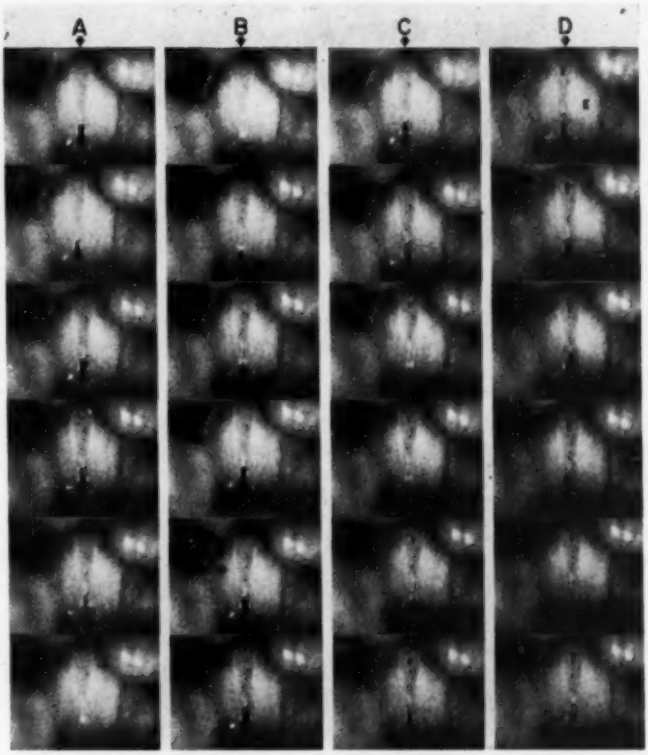

Fig. 7. Damping. In going through the falsetto to its upper reaches, elevation in pitch is produced in some vocalists by approximation of corresponding segments of the vocal cords progressing posteriorly to anteriorly. This sequence shows damped cords opening in descent.

by the other two mechanisms. It is accomplished by progressive closure of the glottis from posterior to anterior as corresponding segments of the vocal cords appose tightly. As the vibrating cordal margins become correspondingly foreshortened, the pitch goes up and is finally reduced to a squeak emitted through a pinpoint opening at the anterior commissure (see Fig. 7). Beyond this the larynx closes in spasm. This type of falsetto action was recognized in the last century and called stop-closure by Mackenzie.[1] It was documented by

the high-speed films of the Bell Telephone Laboratories and termed damping by Pressman.[6] This mechanism is utilized in the high falsetto by some male singers and blends imperceptibly with that portion of the voice above that used in singing. It is, however, by no means a necessary accompaniment of the high falsetto and was seen much less often in this study than the two previously described mechanisms. Most of the male subjects encompassed the entire falsetto with the vocal cords vibrating in their full length, and damping was not seen once in the female. Those male voices employing the falsetto with the greatest skill utilized damping at high frequencies.

There may be more to damping than has been described, because it is impossible to visualize the larynx of certain male vocalists at these very high frequencies. The larynx closes down, and the epiglottis is drawn inward into a covering position as the glottic resonating chamber assumes its smallest possible dimensions. In one male vocalist ascending from middle C to F (f″) one octave and a fifth above, the vocal cords did not dampen posteriorly, but the laryngeal orifice was so contracted anteriorly that they were probably squeezed together in that area.

Stop-closure or damping effectively raises the pitch by a combination of reducing the functioning length of the vocal cords and immobilizing more of their substance. It is presumed to be due to the contraction of controversial interdigitating fibers within the thyroarytenoid muscles, but this remains as yet unproved. Since there is an accompanying narrowing of the larynx in its lateral diameter, the explanation may lie in squeezing of the vocal folds by contraction of more laterally situated muscle bundles.

It must be emphasized that the terms damping and falsetto are not interchangeable. Damping correctly describes only one type of falsetto action and not the major one at that. As such it can no more be used synonymously with the falsetto than can a part be substituted for the whole.

COORDINATION.

The discussion thus far has related to the falsetto break and the falsetto itself. There are some vocalists in whom no break is discernible and whose entire range seems to be encompassed in one register. These voices flow smoothly and evenly from full voice to unmistakeable falsetto with no trace of abrupt change in quality. Sometimes in "dissecting" this type of voice, as it were, for photographic purposes, years of training can be temporarily undone by forcing the subject to vocalize in such a way as to cause the long concealed falsetto break to reappear. Other voices withstand all efforts to reduce them to a more elementary state. These observations are not new, but the mechanism by which a smooth transition is effected has only been hypothecated and not previously demonstrated.

High-speed photography reveals the muscular coordination by which this is accomplished, and the thyroarytenoids are shown to be capable of the most subtle intrinsic adjustments. Starting at either end of the vocal range there is such a gradual change in the appearance of the cordal vibratory pattern that it is necessary to make comparative observations at intervals within the film strip to appreciate them (see Fig. 8). In ascending from the lower register at any one intensity, the vocal folds are first seen striking each other broadly, the entire substance of the thyroarytenoid muscles participating in the vibratory activity. As the pitch rises, they become increasingly taut, but the entire mass of thyroarytenoid is in motion. In the upper reaches of the lower register and in the area of what would be the falsetto break in the incompletely trained voice, the frequency of vibration continues to increase and, in the trained vocalist, there is a decreased activity of the most lateral portions of the thyroarytenoids. Continuing into the highest reaches of the voice and into what is acoustically falsetto, there is no sudden abrupt change in the vibratory pattern. At any given intensity, movement of the lateral aspects of the cords becomes less but does not cease. The glissando scale, or arpeggio is affected so smoothly and with such control that no real transition may be visible.

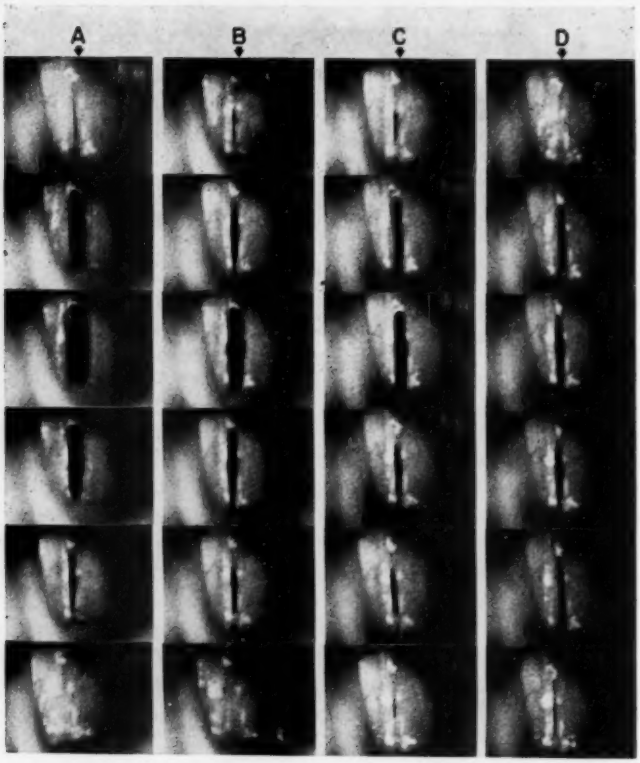

Fig. 8. Coordination. In well trained singers the voice passes from low fundamental registration to falsetto with no abrupt change in quality or appearance of the vocal cords.

In others within the space of a few frames and with such subtlety that it can be easily overlooked, a quick shift from fundamental to falsetto mechanisms occurs. The movements are so well controlled that there is no real "break" and a true blending of registers is achieved. It is clear that in skilled vocalists upward extension to the higher reaches is affected at any given intensity by expert utilization of less thyro-arytenoid substance. The accomplished singer is the complete

master of his thyroarytenoids and achieves the ultimate in delicacy of muscular control.

FACTORS OF PITCH AND VOLUME.

Pitch within the falsetto, just as in the fundamental, is primarily a function of tension of the vocal cords. Lateral X-rays demonstrated little or no elongation of the glottic chink within the falsetto itself. In the trained singer control of the thyroarytenoids is so expert that selective contraction of their fibers will effect elevation in pitch. In the untrained voice, however, there is another and decidedly inefficient factor at work. Control of the medial elements of the thyroarytenoids is so imperfect that they are inadequate for the regulation of pitch. A rise is then accomplished simply by increasing the force of the expiratory air blast, thus forcing the vocal folds to vibrate at a higher frequency even though their state of tension remains unchanged. Pitch may be raised an entire octave by this mechanism alone. Because of excellent muscular control the well-trained singer may swell from pianissimo to fortissimo on the same pitch while the less expert will experience a rise in pitch.

Volume in the falsetto is controlled as in the fundamental register by the force of the tracheal air column. In the incompletely trained voice overblowing in the region of the "break" may cause the falsetto to revert to the lower register by throwing the entire muscle mass into vibration. The trained singer, however, is in such control of his laryngeal musculature that the vocal cords withstand increased air pressure, whatever portion of the thyroarytenoid muscles participating in the vibratory action simply responding by greater lateral excursion. The sound thus produced may be thinner because of a less complex overtone pattern and less powerful because of a smaller mass of moving muscle, but it can still be loud.

TERMINOLOGY.

Although this study has clarified certain features of the

falsetto, it does little to solve the vexing problem of terminology. Laryngologists and voice teachers lament the continued use of such terms as chest register, head resonance, head tones, etc., which have long ago been shown to be without physiologic basis. These terms are bad enough from a scientific standpoint, but more than that, they do not have uniform esthetic connotation within the vocal profession itself. For example, a soprano singing F below high C and, from an acoustical standpoint, utilizing the falsetto, will remark "I don't know what you mean by falsetto. In continuing upward I simply use more head resonance." A second vocalist will then comment, "No, those high tones are obviously falsetto." This is typical of the varying interpretations of a nomenclature in general usage for several hundred years.

It is possible to employ these terms, unscientific as they are, in the untrained voice because cleavages are clearly audible. In the trained voice, however, it is another matter. High-speed films in this study have repeatedly demonstrated that the entire vocal range may consist of only one register in the sense that all tones are produced by the same basic mechanism. Controlled selective contraction and relaxation of the thyroarytenoid muscles will effect a graduated and smooth transition from 100 per cent utilization of the thyroarytenoids in the so-called lower register to a range of perhaps 10 to 60 per cent in falsetto. Using superbly coordinated vocal musculature as raw material the expert vocalist then achieves coloring and further blending by skillful use of the supraglottic resonating chambers. The result is an artistic effect which confounds all efforts to reduce the voice to terms which are descriptive of its primitive stage.

Too many variables are at work for a single terminology to satisfy everyone at this time. It would seem to be more important for greater knowledge and understanding of modern laryngeal physiology to filter through all levels of vocal pedagogy so that there can at least be a meeting of minds on factual ground. If this can be accomplished, nomenclature may well take care of itself, for it will then evolve through a

knowledgeable rather than an enforced substitution of rational terms for those in obvious physiologic and acoustical error.

SUMMARY.

The falsetto refers to the upper part of the singing range situated above the point at which there occurs a change from relatively full and sonorous tones to those of characteristically lighter and thinner quality. The various mechanisms by which this is accomplished are complex but essentially of such a nature that the thyroarytenoid muscles are refrained from vibrating in their entirety as they do in the fundamental register. Three basic patterns of vibratory activity in the falsetto have been identified by high-speed photography. The first is the open-chink, in which the cordal margins do not or only occasionally touch each other in vibration; the second is the closed-chink, in which they do make contact; and the third is damping, in which pitch is raised by progressive approximation of corresponding segments of the vocal cords usually from posterior to anterior. The first two mechanisms are by far the most common and are accomplished with the vocal cords vibrating in their entire length just as in fundamental registration. The distinguishing feature of the falsetto is decreased activity of the laterally situated mass of thyroarytenoid musculature. In damping there is also an actual shortening of the vibrating elements of the cords.

The degree of vocal training is of prime importance in determining both the acoustical qualities of the falsetto and its appearance in high-speed photography. In making transition from the fundamental register to the falsetto in incompletely trained voices, high-speed photography reveals a period of muscular dyscoordination, the length and degree of which is directly related to the vocalist's skill. In the advanced vocalist this so-called "break" is not heard nor is it seen in film sequences, selective contraction and relaxation of the thyroarytenoid muscles effecting a smooth transition from the lower register to the falsetto. This selective control continues throughout the falsetto so that the entire vocal range may

appear to be an extension of one basic mechanism. Variations in vocal skill are associated with such wide differences in acoustical properties of the voice and physiologic behavior of the vocal cords, that it is impossible at this time to encompass vocal registration in a single descriptive terminology.

The findings of this study suggest new areas for experimentation and speculation on the part of the laryngologist, vocal therapist, and voice teacher. The behavior of the vocal mechanism during phonation in the falsetto points, for example, to the possibility of corrective vocal exercises utilizing the falsetto in a comfortable tessitura from piano to mezzo-forte. For the voice teacher there is evidence from this study that it is not only possible but also desirable to develop the falsetto in the normal singing voice, and that a smooth transition from the fundamental to the falsetto register is attainable with proper training.

CONCLUSIONS.

1. A study of the falsetto is reported, based on high-speed motion pictures taken by indirect laryngoscopy at 6,000-8,000 frames per second in vocalists of varying degrees of training and skill.

2. The mechanism of the falsetto is reviewed and observations recorded and interpreted in the light of this photographic evidence.

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PRIMARY ADENOCARCINOMA OF THE UVULA.

Report of a Case.

C. R. SMATHERS, M.D.,
Jacksonville, Fla.,

and

EDMUND VERMES, M.D.,
New York, N. Y.

Primary adenocarcinoma of the uvula is a rarely encountered tumor of the oral cavity. In a 50-year survey of the literature, Stout[1] found no cases of adenocarcinoma in his reported series of 28 tumors of the uvula. New and Childrey[2] reported 357 cases of malignant tumors of the tonsil and pharynx over a 13-year period, 1917-1930, but there was no mention of primary adenocarcinoma of the uvula in this series. In 41 cases of adenocarcinoma of the oral cavity, Watson[3] listed the soft palate as a primary site in three cases. Martin[4] reported 16 adenocarcinomas of the palate, but only four originated from the soft palate. The authors[3,4] did not mention the uvula as a primary site in these soft palate adenocarcinomas. Havens and Ye[5] discussed primary malignant tumors of the uvula, but all seven cases reported were squamous cell carcinoma. Tremble and Lockhart[6] state, "malignant tumors of the soft palate are relatively common; however, those with their primary site of origin in the uvula are rare."

The origin of adenocarcinoma of the uvula is the mucous glands. Glands of the mucous type predominate beneath stratified squamous epithelium; however, beneath pseudostratified ciliated columnar or respiratory epithelium, seromucinous glands predominate.[7] Much confusion has existed between the relation of adenocarcinoma of the true salivary glands and adenocarcinoma of the glands in the upper respiratory tract and oral mucous membrane. This confusion is due to the fact that these neoplasms with similar gland structure

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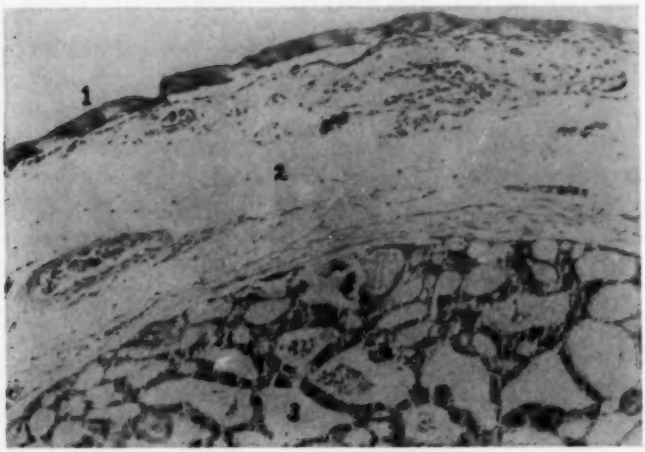

Fig. 1. Low power view of the tumor showing (1) stratified squamous epithelium of the uvula; (2) submucosal layer; (3) adenocarcinoma showing atypical glandular structure.

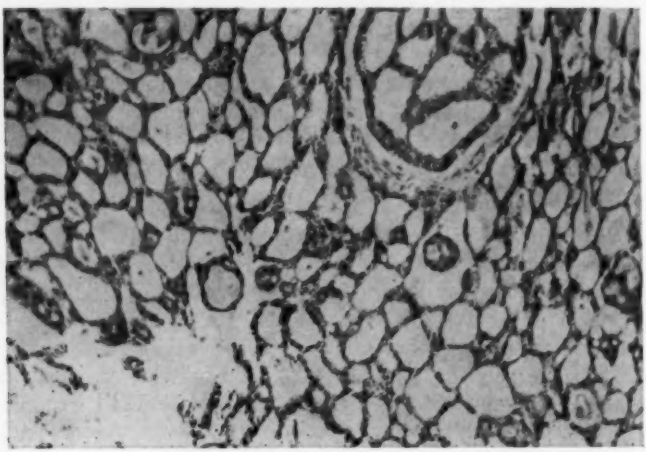

Fig. 2. Low power view illustrating the "Swiss Cheese" type of tumor composed of atypical glands, some of which contain colloid.

may originate from either serous, mucous, or glands of the mixed type. Sharnoff and Lisa[8] call attention to the differentiation that should be made regarding the cylindromas of salivary gland origin and the "true" adenocarcinomas that originate from mucous glands. McDonald and Havens[9] classified 122 cases of adenocarcinoma of the nose, throat and mouth as distinct from cylindromas. They graded these 122 tumors by the Broders method. The authors[9] regarded Grade I and II adenocarcinoma as related to the mixed tumors.

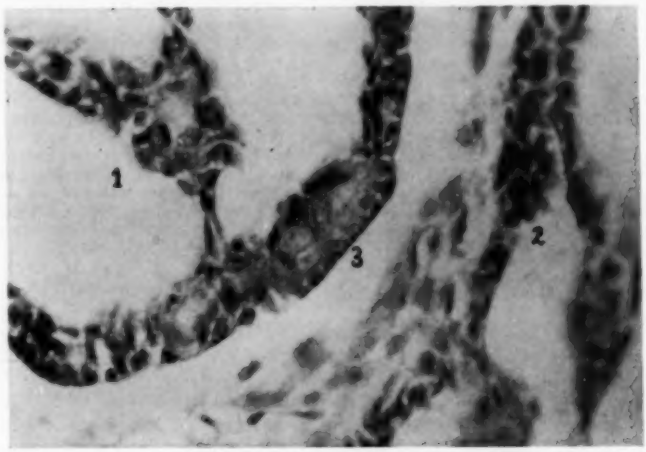

Fig. 3. High power view showing mitosis at (1); hyperchromasia at (2); nucleoli at (3).

Forty-two adenocarcinomas of Grade III and IV in this group were considered to have an origin different from that of cylindromas and papillary adenocarcinoma of the salivary glands. Mixed tumors that originate from the mucous glands and the salivary glands can be distinguished from these "true" adenocarcinomas despite their similar microscopic structure. Mixed salivary gland tumors are composed of epithelial elements, spindle cells, cartilage and myxoid tissue, in addition to the atypical glandular structures present. The acinar

spaces of the atypical glandular structures present in the "true" adenocarcinomas may not be seen in all mixed tumors.

The following case is presented as an example of primary adenocarcinoma originating in the uvula. Such tumors are infrequent, and only a few cases have been reported in the literature.

REPORT OF A CASE.

The patient, a 55-year-old white male, had noticed a slowly growing "lump" on his uvula for four months. No other symptoms were present. Laboratory studies were within normal limits. The patient was advised to have the tumor removed without further delay. On May 3, 1956, the entire tumor was removed under general anesthesia without incident. The patient was discharged the following day. The tumor measured 1.5x1x1 cm. on gross examination. Pathology report by Dr. Andrew A. Eggston was as follows: Microscopic sections reveal a surface of stratified squamous epithelium which has an ulcerated area, around which some lymphatic inflammatory reaction is present; some mucous glands surrounded by inflammatory reaction as well as trabeculae of collagenous fibrous tissue are also present. The outstanding features are seromucinous glands that show marked cellular proliferation. The cells form atypical glandular structures and show anisocytosis, hyperchromasia and mitosis. The entire structure is that of cylindromatous or "Swiss Cheese" type of adenocarcinoma. Diagnosis: Adenocarcinoma of the Mixed Seromucinous Gland type.

The above case is asymptomatic and free of disease over three years after surgical removal of the tumor.

COMMENT.

Primary malignant tumors in the uvula are rare. Otolaryngologists performing tonsillectomies have an excellent opportunity for routine examination and palpation of the uvula. Histological examination should be performed in all cases where the uvula may be removed for any reason. The functions of the uvula were described by Richardson and Pullen.[10] They considered the uvula of more importance in deglutition than in speech. As illustrated by the above case, removal of the uvula apparently does not seriously impair the swallowing function.

SUMMARY.

A discussion of adenocarcinoma of the uvula is presented, together with a selected review of the literature. The relative

rarity of these tumors is emphasized. One additional case is presented.

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1842 King street.
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RECOGNITION AWARDS.

Hans von Leden, M.D., and Paul Moore, Ph.D., of Northwestern University recently received the Gold Medal of the Illinois State Medical Society and the Hektoen (Bronze) Award of the American Medical Association for original investigation. These Awards were granted in recognition of their research on the mechanics of the crico-arytenoid joint.

THE CROSSBAR (POLYETHYLENE) PROSTHESIS FOR THE IMPROVEMENT OF HEARING.

Initial Report.*†

G. DOUGLAS HAYDEN, M.D.,

Richmond, Va.

To accomplish the objective of a tympanoplasty procedure, various techniques have been described since 1950. Type IV tympanoplasties have consistently given the poorest results in hearing improvement. Type IV tympanoplasty, as described by Wullstein,[1] is where erosion of the crura of the stapes has occurred, leaving only an intact mobile footplate. The tympanic membrane or graft can not be brought into contact with the footplate for sound transformation to occur as in a Type III tympanoplasty in which a healthy mobile stapes is present.

Wullstein's technique in Type IV tympanoplasty has been to leave the mobile footplate exposed directly to the sound waves while protecting the round window with a graft. This procedure is based on the physiological principle that there must be a difference in the time and phase relationship by the two windows for stimulation of the cochlea. Besides the technical difficulties in maintaining this difference between the two windows, the end result of improved hearing left much to be desired. In trying to improve his results, Wullstein has reported the use of an oval ring made of "Polavit" fitting in the oval window niche between the mobile footplate and tympanic graft acting as an acoustic transmitter. Apparently, these results were not too encouraging.

Since then, Shambaugh[2] and Guilford[3] have reported improved results with the use of a polyethylene prosthesis be-

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*Presented at a meeting of the Tidewater Ophthalmological and Otolaryngological Society, Portsmouth, Va., May 25, 1960.

†From the Department of Otology, Rhinology and Laryngology, Medical College of Virginia.

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tween and drum or graft and footplate. The prosthesis consisted of a polyethylene strut with a flared end. In using this single strut I found it difficult to maintain its correct position. The strut can easily slip out of position because of the difficulty in maintaining a tight connection between the bony footplate and the soft tissue of the graft. Some of the oval window niches are quite shallow and the strut can slip

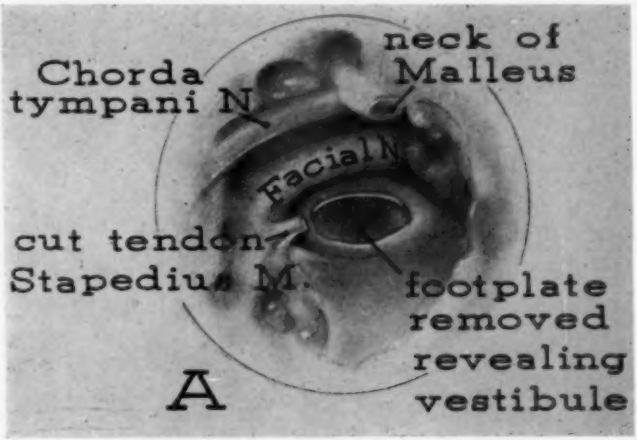

Fig. 1.

out of position easier than with a deep niche. The tight fit necessary to maintain the proper position between the graft and bony footplate makes extrusion of the strut through the graft a very likely possibility.

To overcome these difficulties, I have been using the following procedure with gratifying results in "Type IV" tympanoplasty technique:

After completing all the necessary removal of infected tissue and diseased bone, the oval window niche and footplate are treated exactly as in performing a Shea[4] fenestration of the oval window. The promontory is saucerized if necessary and the footplate of the stapes is removed, even if it is mobile

(see Fig. 1). A flattened piece of gelfoam (or a vein graft) is placed over the oval window. A previously-prepared crossbar polyethylene prosthesis (see Fig. 2) is then inserted between the oval window and graft. The crossbar consists of a No. 50 polyethylene strut with one end flared and the oval window end slightly tapered, going very loosely through a

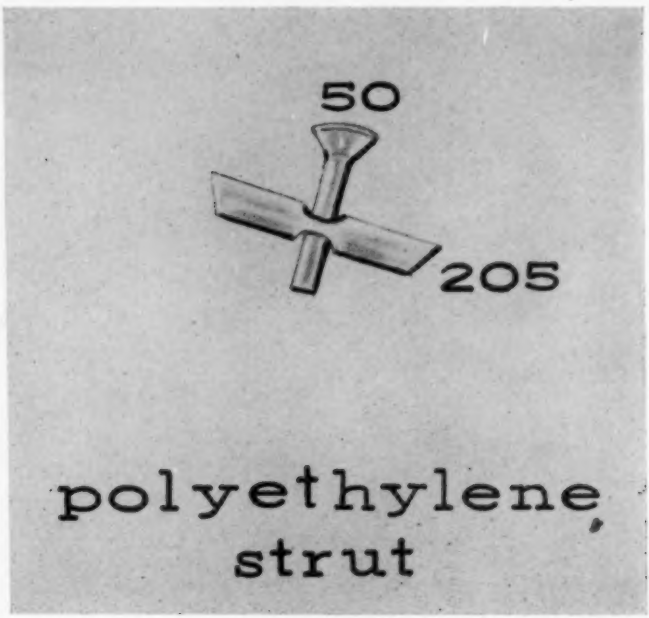

Fig. 2.

piece of No. 205 polyethylene tubing (see Figs. 3 and 4). The No. 205 tubing acts as a stabilizing influence and keeps the acoustic transmitter (No. 50) in place.

Removal of the footplate, even if it is mobile, makes for a much better maintenance of position of the prosthesis, and improved hearing results because of better motility instead of a more static condition as in the Wullstein oval ring of Polavit.

The No. 50 polyethylene strut is not likely to extrude unless one makes it too high.

The size of the crossbar strut can be varied. When there is no bridge, as in radical cavities, the No. 50 strut should be slightly shorter than in operations in which the bridge is preserved. If the strut is too high it tends to cause atrophy and perforation of the drum and graft.

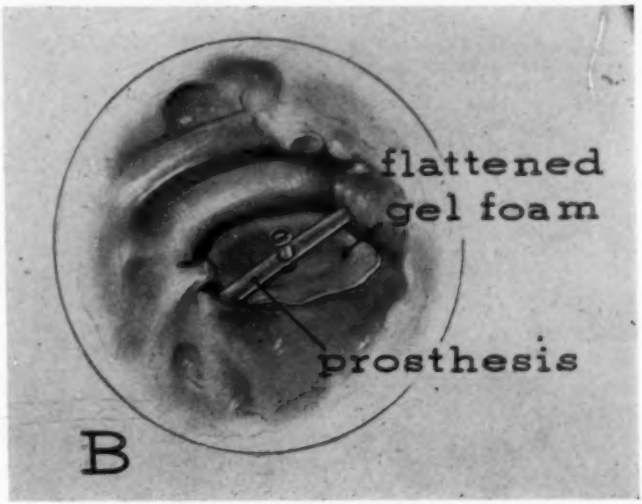

Fig. 3.

The same prosthesis has been used in fenestration of the semicircular horizontal canal failures with success. The improved hearing in every case has been as good or better than a regular revision of the fenestrated canal could hope for.

TECHNIQUE.

A good-sized flap of skin is elevated in the fenestrated cavity and the stapes exposed. A fenestration of the oval window procedure is then performed. A flattened piece of

gelfoam is placed over the vestibule and a crossbar prosthesis is fitted into position between the tympanic membrane and the oval window opening, supported by the flattened gelfoam. In an occasional case, one may find it necessary to use a small skin graft if the flap is too fragile to elevate properly making it impossible to close the middle ear without it. I have had encouraging results with seven straight cases in the past six months using this method.

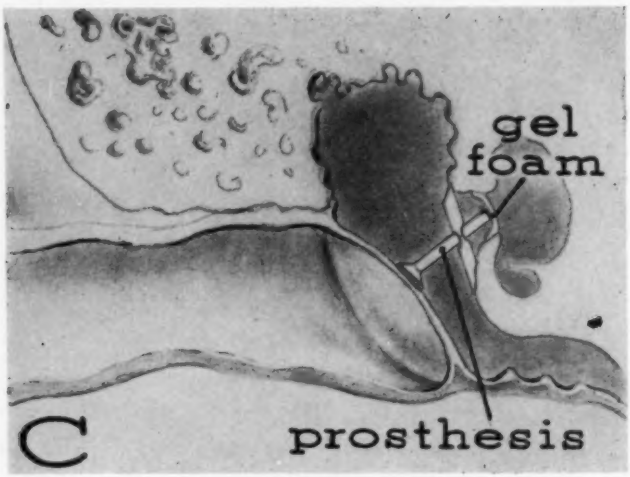

Fig. 4.

SUMMARY.

A polyethylene prosthesis with a stabilizing bar can act as a good transmitter for the improvement of hearing in Type IV tympanoplasties. The maintenance of its position and the improvement of hearing can be enhanced by removal of the footplate even when mobile.

The same type of prosthesis has also proved successful in improving the hearing in certain patients who have previously undergone a fenestration of the semicircular horizontal canal

and who still have a good cochlear nerve response but who failed to regain or maintain useful hearing.

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ESOPHAGEAL NODULE OF THYROID TISSUE.*†

GABRIEL PÔRTO, M.D., F.I.C.S.

Campinas, Brasil.

The paper describes an extremely rare case and is only the second one published in the medical literature.

CASE REPORT.

A 56-year-old male peasant, living in São João do Itaguacu, Estado de São Paulo, Brasil, came for consultation, March 31, 1959.

The patient told me that one month prior to this visit he had felt a little pain on swallowing. Nose, mouth, pharynx and larynx were normal. The thyroid gland was not enlarged; in fact, it was difficult to locate by palpation. X-ray examination showed a normal esophagus.

Esophagoscopy was done on April 1 using Jackson's tube, 7 mm., and by this means a tumor was discovered attached to the midline of the cervical esophagus. The tumor was bleeding, and, after aspiration of the blood, was clearly seen. It was about the size of a pea; moriform in contour, suggesting an esophageal papilloma. By means of a forceps it was easy to remove the small tumor entirely for biopsy.

The histo-pathologic examination was made by Dr. Monteiro Salles, and his report was: ectopic nodule of the thyroid gland.

Functional test of the thyroid gland by I^{131} (Isotopo Diagnosis) was made by Dr. Ubajara C. P. Correa, April 8, 1959. His conclusion was hypofunction of the thyroid gland according to the basal metabolism. Lugol solution, thyroid extract, mineral salts, vitamins, especially vitamin B^{12} were prescribed by Dr. José Estevan Teixeira de Camargo, April 8, 1959.

The patient returned to the office, June 11, 1959, having recovered from the esophageal symptoms. On the following day an esophagoscopy was done; no abnormality of the esophagus was observed.

COMMENTS.

This case can be named an esophageal ectopic thyroid or an esophageal aberrant thyroid as both denominations mean the same condition. I prefer esophageal nodule of the thyroid, because thyroid tissue found in the esophagus obviously must be aberrant or ectopic.

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*Presented at the VIII Meeting of the Brazilian Society of Oto-rhino-laryngology and Bronchoesophagology in Porto Alegre, Sept., 1959.

†From the Department of Otolaryngology, Instituto Penido Burnier, Campinas, Brasil.

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The present observation is extremely rare; it is the second case reported in the medical literature, the first case having been presented by Lawson Whale, laryngologist to the Hampstead General and the London Temperance Hospitals and was published in the *British Medical Journal*, 2:987, Dec. 10, 1921.

Studying the embryology of the thyroid gland one can realize the genesis of these cases. The thyroid gland is formed very early in the human embryo (with 18 somites only), before the development of the neck. In this phase of

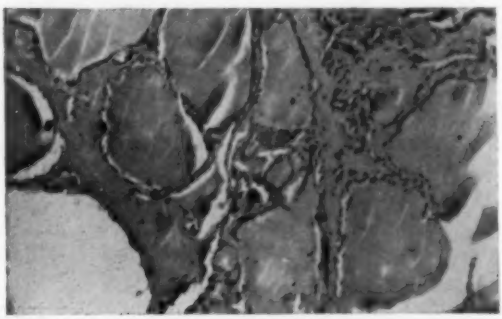

Fig. 1. Photomicrograph showing the thyroid tissue found in esophagus (X100).

evolution the thyroglossal duct is related to the heart, arterial trunk and the sketch of the esophagus. Not until the seventh week does the neck begin to assume its structure.

In some aspects our case resembles Whale's observation. The tumor in both cases was seen as a benign lesion, like an esophageal papilloma. I believe that frequently these thyroid nodules can be infected. In Whale's case the infection process was serious. The symptomatology presented by his patient suggested to him the possibility of esophagus perforation and infective mediastinitis because the patient told him that she had swallowed a fish-bone. In my case the symptoms of infection were not so serious, but they were evident and were revealed by the odinophagy and the bleeding of the aberrant

thyroid. These cases usually have no symptoms; they can be discovered only when the thyroid tissue is infected.

In both cases reported in the literature the treatment was easily carried out and the small thyroid nodules were completely removed through the esophagoscope.

SUMMARY.

One case of esophageal nodule of thyroid is reported. The tumor was found in the esophagus cervical and was completely removed through the esophagoscope. This observation is extremely rare; it is the second case published in the medical world literature . The first one was presented by Lawson Whale in the *British Medical Journal* (2:987, Dec. 10, 1921).

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Annual Meeting: October 8, 1960, Belmont Hotel, Chicago, Ill.

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Meeting:

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Meeting: Oglethorpe Hotel, Wilmington Island, Savannah, Ga., March 2-4, 1961.

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Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:30 P.M. last Monday of each month from September to June, inclusive—Otolaryngology Section. 6:30, first Thursday of each month from September to June, inclusive—Ophthalmology Section.

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Meeting: Edgewater Gulf Hotel, Edgewater Park, Miss., May 12-13, 1961.

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Meeting: Second Tuesday in each month at 8:00 P.M. at Memphis Eye, Nose and Throat Hospital.

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Time and Place:

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Meeting: Jointly with the North Carolina Eye, Ear, Nose and Throat Society in Greenboro, N. C. Headquarters will be in the King Cotton Hotel, Sept. 13-15, 1960.

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Meeting: St. Louis, Mo., October 31, 1960, to November 3, 1960.

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